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A handbook of skin conditions in Aboriginal populations of Australia

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FRONT COVER
(Left) Residual ochre
(Centre) Ringworm on the body due to *Trichophyton rubrum* (granular variant)
(Right) Pityriasis versicolor

FOREWORD

I am very pleased to be able to support the production of this book. We are committed to improving access to and quality of primary health care services for Aboriginal and Torres Strait Islander people and I believe this book will fill an important niche in this process.

Dr Allen Green is a Dermatologist who devoted much of his career to visiting remote communities throughout Northern Australia providing dermatological services. Over a period of three decades he learnt about the skin diseases that affected Aboriginal Australians and worked closely and collaboratively with Aboriginal communities in devising effective treatment programmes.

Dr Green has presented his findings from time to time at professional conferences throughout Australia and has had a number of his observations published in medical journals. In 1997 the World Congress of Dermatology was held in Sydney and at this meeting Dr Green was encouraged to present a summary of his life's work. As a consequence, he was awarded the Certificate of Appreciation of the International League of Dermatological Societies, one of the highest awards in the International Dermatology community for his contributions to the health of Aboriginal communities.

I viewed the display of Dr Green's work at the time of the Congress and was struck by the unique value of this work and the need for it to be preserved. Moreover it was clearly a valuable resource for dermatologists and other health professionals in the ongoing effort to deliver high quality health services to Aboriginal Australians.

I commend all those involved for their dedication and efforts in preparing this book, particularly Dr Delwyn Dyall-Smith and Dr Alan Cooper of the Australian Dermatology Research and Education Foundation whose individual efforts have made this possible. In achieving this, you have been able to ensure the great body of work and knowledge compiled during Dr Green's career is preserved and utilised by generations to come. In a field where we continue to address serious health inequalities and great challenges we must welcome and use all experience and knowledge and ensure its dissemination to a wide audience. This book is an important contribution to this ongoing effort. I especially thank and commend Dr Green for his work

and dedication throughout his career to this most important endeavour and for agreeing to share this experience and effort. I trust that this will lead to improved health care and outcomes in Aboriginal health and I hope that we can look forward to a time when much in this book will be of historical record only.

Dr Michael Wooldridge
Minister for Health and Aged Care

FOREWORD

We are pleased to have had the opportunity to contribute to the development of this handbook for use by health professionals around Australia. The National Aboriginal Community Controlled Health Organisation (NACCHO) is the peak Aboriginal health body in Australia and represents over 100 Aboriginal Community Controlled Health Services. Our sector is a considerable intellectual resource on Aboriginal health matters, enabling us to deliver appropriate care and to advocate effectively for Aboriginal people in matters of health.

Through our advisory panel, earlier drafts were critically appraised, summaries were included to aid health workers, and decisions on cultural issues were made weighing up the benefit of this book as an educational tool against the harm that could arise if the book revealed sensitive cultural practices.

As a result of our collaboration with the authors, we believe this handbook has significantly benefited from our efforts and could be useful Australia-wide. Many health professionals need the information included in this handbook in order to make better clinical decisions. The pictures make it easy for people working in Aboriginal health to identify some of the skin conditions and diseases that are suffered by our people.

Whilst this handbook will help health professionals to make a diagnosis, we encourage these health providers to understand that skin diseases can result from poor living conditions. Some of the skin conditions that you will see in this book are endemic in Aboriginal communities and contribute to our reduced life expectancy. Our situation is not unlike that in many developing countries. The 1996 Australian Bureau of Statistics census reported that 90% of the Australian households living in severely overcrowded conditions (more than 12 people living in a three-bedroom house) were Aboriginal.

The solutions to diseases of poverty are to be found in improved community infrastructure and Aboriginal community control over matters of health. The principle of Aboriginal self-determination is central to our health. We see health as not just individual physical well-being but the social, emotional and cultural well-being of the whole community. Our well-being is intrinsically land-centred. Health care to us should be delivered through our full participation at every stage in the spirit of self-reliance and self-determination. It contrasts with the view that the health of people is the domain of specific agencies or professionals with the tendency to break the

body up into little parts. Our approach is holistic. That's why I have been saying that *in the implementation* of health care to Aboriginal peoples, the 'organised' approach, not the 'organ' approach, is needed.

We need health professionals to work with us in advocating for government policy that will address the environmental and socio-economic determinants of health and reduce the health inequity of Aboriginal people. We all know that the factors that determine health are largely outside the control of the health sector.

No health professional can ever 'do' Aboriginal health. If you want to be involved in Aboriginal health, my best advice to you is to involve Aboriginal people in a true partnership from the very beginning. I hope this book is of some value in helping our people and I look forward to seeing you in the future.

Puggy Hunter
Chairperson of NACCHO

CONTENTS

Dr Allen C Green

MB BS DDM DTM&H DPH FRACMA FACD

Allen Green, a Tasmanian by birth, graduated in medicine from the University of Melbourne in 1947 (MB BS), then worked as a resident medical officer at the Royal Melbourne Hospital.

In 1950, he assisted a prominent Melbourne dermatologist, the late Dr Ivan Wartzki, leading him to gain the Diploma of Dermatological Medicine (DDM) at the University of Sydney in 1956. He spent the next year in Europe and the United Kingdom, gaining further experience in dermatology, including at St John's Hospital for Diseases of the Skin, in London.

In 1960, Dr Green joined the Commonwealth Department of Health. He went to Darwin as a general medical and quarantine officer and flew with the Northern Territory Aerial Medical Service, developing an interest in skin conditions among Aboriginal Australians. Allen rapidly rose from a base grade medical and quarantine officer in Sydney, Darwin, Perth and Canberra. In 1965 he took over administration of the Northern Territory Medical Service in Darwin where he continued his interest in Aboriginal health. His last post was Commonwealth Director of Health for South Australia in November 1968. He retired from this position in 1981.

While working as Assistant Director General (Public Health) he served on numerous committees of the National Health and Medical Research Council and the Public Health Advisory Committee.

He obtained further post-graduate qualifications, gaining the Diploma of Tropical Medicine and Hygiene in 1962 (DTM&H), Diploma of Public Health in 1966 (DPH), Fellowship of the Royal Australian College of Medical Administrators in 1968 (FRACMA) and Fellowship of the Australasian College of Dermatologists in 1969 (FACD).

While working as the Commonwealth Director of Health for South Australia, Allen became an associate visiting dermatologist at the Adelaide Children's Hospital from 1969 until 1983. He visited the Northern Territory periodically when no dermatologist was

Allen Green on return from a bush trip.

there, continuing his interest in skin conditions among the Aboriginal population.

After retiring from the Commonwealth Department of Health, he started in private dermatology practice in Mildura, Victoria, continuing until February 1997 when ill health forced him to reluctantly retire.

Allen commenced the delivery of dermatological services to Aboriginal populations in 1960. He worked all over Australia and the Torres Strait Islands, and many remote parts of Australia.

At the 19th International Congress of Dermatology, held in Sydney from 15–21 June 1997, Allen presented a photographic and descriptive exhibit entitled 'Skin Conditions Among the Australian Aborigines'. This attracted great interest among dermatologists from all over the world. The presentation included over 600 photographs, numerous panels of information, Aboriginal music and art.

Allen's international and national reputation for his dermatological work among Aboriginal Australians was recognised at this Congress when the committee of the International League of Dermatological Societies gave him one of its highest awards for his dermatological work among the Aborigines – the Certificate of Appreciation.

Some of his friends thought of Allen Green as 'the bush dermatologist'. He deserved and enjoyed the title.

Section 1

Introduction to Dermatology

SKIN CONDITIONS AND SKIN DISEASES

What is a skin condition? It can mean many things: a normal mark of genetic origin; a cosmetic blemish with or without hope of removal; obligatory injuries of social importance in one's inherited or acquired culture; the results of an individual's environmental hazards; or a skin disease.

And what is a skin disease? In the past, some diseases with striking signs in the skin were called 'skin' diseases. Other diseases, some showing prominent signs in the skin, were classed as internal (or systemic) diseases. Fewer 'skin' diseases are now being regarded as belonging to the skin alone. Many signs in the skin are pointers to internal disease. The changes in skin of patients with acquired immunodeficiency syndrome (AIDS), a systemic viral infection, is a contemporary example.

What is seen in the skin results from many influences. These include:

- Genetic and development;
- Environmental – natural, man-made;
- Physiological;
- Cultural beliefs, customs, practices;
- Causes of disease;
- Pathological processes – causes being either known or unknown.

Such influences result in structural and functional changes in the skin. These changes determine the symptoms and signs seen in skin conditions and 'skin diseases'.

For reasons such as these, sharp divisions between 'skin' diseases are neither practical nor rational. Not so many years ago, there were skin specialists who fancied that their work was removed from general medicine and environmental considerations. Nowadays such opinions are not valid.

Dermatology means the study of the various combinations and relationships between the causes of skin conditions, skin diseases and systemic diseases.

Dermatology should be approached and practised with the general principles of medicine and surgery in mind, remembering that 'nature is neither husk nor kernel; She is all in one'. Not to do so is to miss the substance of dermatology.

STRUCTURES AND FUNCTIONS OF THE SKIN

Common structural units of both dark and light skins are:

- Epidermis
- Dermis
- Skin appendages – hair, nails, pilo-sebaceous follicles, eccrine and apocrine sweat glands
- Subcutaneous fat.

The number and density of melanocytes in dark and light skins are similar in comparable areas.

Dark and light skins have similar functions. These can be considered as:

- A *container* for the body with physical properties – strong, tough, waterproof, elastic, compressible and expandable.
- A *protector* against environmental hazards. These include physical influences such as heat, cold, ultra-violet radiation, electricity; chemicals; vitamin D deficiency; and biological causes of disease.
- A *regulator* of body temperature, fluid and electrolyte balance.
- An *indicator* of health and disease. The general appearance of a person, well or ill, largely depends on the condition and an assessment of the skin, its appendages and functions. We say 'You look well/ill'.

Mechanisms subserving the structures and functions of skin

These include:

- Epidermal replacement by the multiplication of cells in the basal layer;
- Development of keratinocytes and keratinisation;
- Formation of the 'acid-mantle' from scales, sebum and sweat;
- Circulation;
- Sweating;
- Sensation;
- Synthesis of vitamin D;
- Inflammatory responses.

There is no evidence that supports any difference in structure or function of the skin between Caucasian and Aboriginal skin types.

When various influences, be they genetic, developmental, physical or biological, impair the structural stability and functional capacity of the skin and their subservient mechanisms, skin damage and disease result.

DIFFERENCES BETWEEN DARK AND LIGHT SKIN

Conditions in dark skins often look quite different from similar ones in light skins. Some of these differences depend on:

- Colour;
- Blood flow;
- Responses to injuries, infections and various pathological processes;
- External matter.

Aboriginality is not determined by the colour of the skin, but is a cultural asset.

Colour

The colour of the skin and its variations in health and disease are determined by various influences including heredity, natural pigments such as melanin, blood (oxygenated and reduced haemoglobin, methaemoglobin) and the bile pigments (bilirubin) and carotene.

There are ranges of colour between groups and individuals and in different body sites in the one individual, some of which are due to influences such as sun exposure, pregnancy and certain drugs. The thickness of the epidermis, especially of the horny layer (stratum corneum), stretching of the skin, and the absorption and reflectance of light are physical influences on skin colour.

Colour is important in the examination and assessment of changes in the skin seen in systemic and cutaneous disorders.

Colour changes may be localised or generalised. Examples include pallor in anaemia and hypochromic naevi; redness in rosacea, sunburn, sun-damaged skin and many inflammatory conditions such as psoriasis and exfoliative dermatitis; yellow in jaundice and xanthomata; lemon-yellow in pernicious anaemia; yellowish-red in pityriasis rubra pilaris and orange in carotenaemia. There are shades of brown in sun-tanned skins, the 'mask of pregnancy' (melasma/chloasma) and from the use of some oral contraceptives; and shades of violet in cyanosis, methaemoglobinaemia and lichen planus. Mongolian spots have a slatey-grey colour (discussed further on page 92), as do some people with haemochromatosis. Albinos have a pink or near-white skin. Similar colour changes are seen in halo naevi and in patches of vitiligo. Browns and blacks are seen in melanocytic naevi (moles) and melanomas. Bruises and purpuric spots go through a series of colour changes from purple, red, green to yellow before fading. Those with sea or airsickness sometimes look green, as did Victorian ladies with chlorosis – a condition no longer seen.

Decreases in melanin

Loss of skin colour resulting from decreased melanin is more obvious in dark skins than in light. This is especially so when the loss of pigment is complete (depigmentation, achromia) as in vitiligo or halo naevus (discussed further on page 90). The affected areas make a sharp contrast against the normal skin.

In people with dark skins, loss of colour can cause cultural, social and personal problems as well as diagnostic difficulties. This is particularly true where leprosy is endemic. An early sign of that disease may be an area of skin showing loss of colour.

People with dark skin who experience localised or generalised areas of decreased or absent skin colour can be expected to be greatly concerned. They should be given a prompt and precise diagnosis and sympathetic consideration by medical and nursing personnel. Patients with dark skin should be warned that they may develop areas of decreased or lost skin colour after certain injuries and in the course of some skin conditions.

Skin colour may be lost after cryotherapy with dry ice or liquid nitrogen, electrocoagulation or cauterisation. If such treatments are proposed, patients with dark skin should be first warned about the possible loss of skin colour (discussed further on page 100).

Increases in melanin

Darkening of the skin (hyperpigmentation) commonly results from increases in melanin.

In naturally dark-skinned people, the skin gradually becomes darker in the first year or two after birth. Skin exposed to the sun can be sunburned and become darker.

For Aboriginal Australians and other people with dark skin, Mongolian spots are normal and represent deep dermal melanocytes (discussed further on page 92). Pigmented moles (melanocytic naevi) of different kinds also represent localised collections of melanocytes, usually in the epidermis or superficial dermis.

Pregnancy and the use of oral contraceptives can result in increased pigmentation of the face (melasma/chloasma) in some women. Regardless of the natural skin colour, most females dislike a patchy darkening of their facial skin. However, some women with light skins aspire to an evenly sun-tanned face and body. Pregnancy also results in darkening of the nipples, areolae, the external genitalia and adjacent skin on the upper thighs. A dark line (linea nigra) develops between the pubic region and the navel (umbilicus) on the lower abdomen. Such pigment changes are normal in pregnancy. They occur

in females with light or dark skin, but are usually more evident in the latter. In dark-skinned people and in brunettes with dark skin, the pigmentary changes in the nipples and areolae are permanent to a greater or lesser degree.

Pseudo-acanthosis nigricans (discussed further on page 94) and dermatosis papulosa nigra (discussed further on page 32) show localised increases in melanin pigmentation. These two conditions are common in Aboriginal Australians.

Skin sites affected by injuries, burns, infections and inflammatory diseases may become darker or lighter (discussed earlier). In the former, the increase in colour is due to pigment incontinence; the epidermal melanin falls down into the dermis and is collected in macrophages (melanophages) (discussed further on page 100).

Changes due to other pigments

Altered blood pigments, bile pigments and excessive amounts of carotenes can result in various colour changes in the skin. Usually these are evident in light skins but may be easily missed in dark skins.

As the palms and soles are usually lighter in Aborigines, these areas should be examined to assess changes in colour, as should the sclerae and the mucous membranes of the eyes, mouth and sometimes the ano-genital region.

Signs such as the yellow skin in jaundice are usually masked by a dark skin and pigmented sclerae.

Alterations in blood flow

Increased blood flow to the skin occurs in various inflammatory skin conditions, common febrile illnesses (e.g. measles) and some systemic diseases (e.g. thyrotoxicosis). The resulting redness, be it localised as in macules (flat) or papules (raised) or generalised, is easily seen in light skins. In dark skin areas of increased blood flow are darker, even violaceous, so that redness is not an accurate description.

Some localised increased blood flow, for instance cellulitis, may be better appreciated by the heat felt when touched rather than by the colour change.

Macules and papules related to increased blood flow appear darker than the unaffected skin. They partly fade on pressure applied with a piece of clear plastic such as a plastic ruler (diascopy, see page 17). The macules of measles and secondary syphilis and the rose spots of typhoid fever can be difficult to see and are easily missed in dark skin.

Bruises (ecchymoses) and pin-point spots of bleeding (purpura) result from major and minute outflows of blood from blood vessels, respectively. They are

not easily seen in dark skin but occur as areas slightly darker than unaffected skin. They do not fade on diascopy. Bruises may overlie damage to soft tissues and bones. Purpura due to blood vessel inflammation (vasculitis) are palpable.

Weals (welts) in dark skin are usually lighter in colour than the surrounding skin. In pale skin these lesions are much more easily seen.

Responses to injuries, infections and pathological processes

Injuries such as abrasions and lacerations, bites and stings, burns, and infestations such as scabies and head lice, frequently become secondarily infected in poor socio-economic living conditions. Crusted ecthymatous sores are common sequelae. They heal leaving scars that are often hyperpigmented.

Neglect, improper management and secondary infection can result in gross changes in the clinical appearances and obscure the underlying condition.

Trichophyton rubrum (granular variant) is the cause of endemic ringworm (tinea) in the high rainfall regions of northern Australia (discussed further on page 56). Very common in many Aboriginal communities, this fungal infection produces a chronic inflammatory response. It is less common in the more temperate climate regions of Australia and in non-Aboriginal people, but when it does occur infection with this dermatophyte results in appreciable redness of the affected skin.

Syphilis, the prevalence of which is reducing in Aboriginal populations, may produce secondary lesions such as exuberant condylomata lata. These days florid secondary syphilis is unusual. This is perhaps partly a result of the widespread use of antibiotics for other conditions but effective against syphilis (happenstance antibiotics). In those immunologically compromised the clinical signs of infections can be particularly striking.

Discoid lupus erythematosus (DLE) appears to be much more common in Aboriginal than in non-Aboriginal people (discussed further on pages 35, 48 and 66). This applies particularly to DLE on the lips. In dark skins many of the appearances of DLE are more striking and squamous cell carcinoma (SCC) can complicate DLE, especially on the lower lips.

Sun-related skin cancers, as seen in Caucasians with light skins, particularly those of Celtic origin, are practically unknown among Aboriginal Australians. Melanoma occurs on the foot but is a rarity. No Aboriginal cases were reported in the Queensland Melanoma Survey. Squamous cell carcinoma may, however, affect the pinna (ear) and perianal skin (Dr John Fraser, pers. comm., 2000).

Scales are often more easily seen in dark-skinned people. This is due to the colour contrast between the scales, which are lighter in colour than the dark skin from which they arise. Scales on dark skin vary from whitish to grey to shades of brown. Examples include the white handkerchief form of pityriasis versicolor (discussed further on page 78), the superficial scaling form of *T. rubrum* (granular variant) (discussed further on page 80) and crusted (Norwegian) scabies (discussed further on page 83). The fine scaling seen after measles is more evident on dark skin than light.

Epidermal thickening can partly mask the colour of melanin. On the palms and soles, calluses and warts have a yellowish colour (discussed further on page 126).

Keloids and hypertrophic scars are common reactions in people with dark skin (discussed further on page 110). Keloids over joints are often disabling and difficult to manage.

External matter

Dust, sand, mud, ochres, clays, fats and oils used in ceremonies can result in curious patterns on the skin and scalp (discussed further on pages 52, 74).

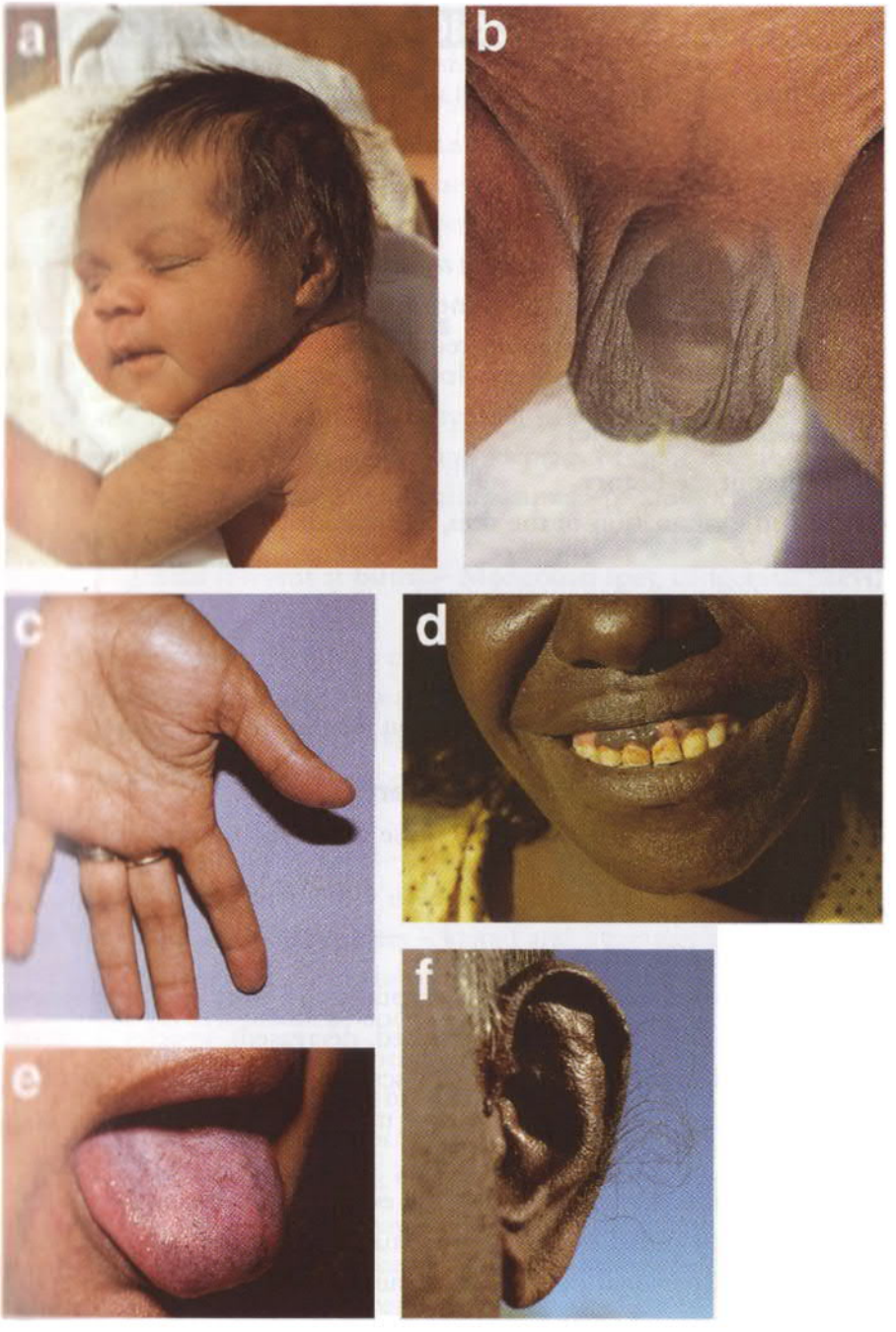
Tattoos of various kinds produced by different materials and methods are common in Aboriginal children, adolescents and adults.

Conditions unusual, rare or apparently absent in Australian Aborigines

These include alopecia areata, atopic dermatitis, dermatomyositis, generalised morphoea, granuloma annulare, lichen planus, pemphigus, pityriasis lichenoides, pityriasis rosea, pityriasis rubra pilaris, psoriasis, sarcoidosis, scleroderma, infantile haemangioma (strawberry naevi), stucco keratoses, sun-related skin cancers (except on the pinna, peri-anal skin and on the lip in DLE), varicose veins and related conditions such as venous stasis dermatitis and varicose ulceration.

Subsequent experience may change this list.

The apparent absence or rarity of these conditions, especially psoriasis and atopic dermatitis, and the frequency of others such as DLE and the liability to keloids, remain challenges for future Australian dermatological research.



a *The newborn Aborigine, the skin is generally pale.*
b *Hyperpigmentation of the male genitalia in a newborn Aboriginal infant.*
c *Hyperpigmentation of the palmar creases.*
d *Pigmentation of the gums.*
e *Pigmentation of the tongue.*
f *Hairy ears – sparse.*

COURSES IN DERMATOLOGICAL CONSULTATIONS

When consulted by a patient with a skin complaint, a doctor can take several courses.

1 Make a preliminary assessment
2 Consider the diagnosis as:
  2.1 Correct on clinical grounds
  2.2 Probable – simple confirmation needed
  2.3 Possible – further investigations required
  2.4 Not possible

  If a diagnosis is not possible:

3 Take an appropriate history
4 Do a detailed examination of the skin, its appendages, mucous membranes
5 Reconsider the diagnosis
6 To treat or not to treat?
7 Get another opinion – or carry on?

It is very important to record identification details for each patient.

1 Make a preliminary assessment

First ask where the present skin trouble is, the duration and main symptoms of the complaint (such as itch and pain).

Ask the patient to undress.

Then inspect the affected areas.

Observe the general features of the eruption and note the component lesions (changes in colour (different, increased, decreased), vesicles (blisters), papules (lumps and bumps), etc.). Signs of scratching, rubbing or infection may be evident. These are important as they may call for particular forms of management.

During this preliminary assessment of the eruption, also assess the patient. Points to be quickly noted include group; nationality; age; gender; understanding of English; personal care and hygiene (clean clothes, body, hair and nails or unkempt, unwashed and smelly); evidence of excesses in tobacco, alcohol or other drugs; obesity or thinness; and occupation (indoor or outdoor worker).

The general appearance of a patient is often a valuable guide to diagnosis. A moment's reflection will show that changes in the structure, functions and colour of the skin largely determine the general appearance. Add what may

be seen in the eyes, the hands, the speech and the gait. All manner of clues to medical and dermatological diagnosis may be there to see. Consider but a few:

- The lax, dry skin associated with recent weight loss and dehydration.
- The warm, moist skin, tremulous hands and the eye signs of thyrotoxicosis.
- The lemon-yellow pallor frequently associated with pernicious anaemia.
- The suffused, high colour in polycythaemia and some alcoholics.
- The yellow tinge in the eyes with jaundice.

2 Consider the diagnosis

2.1 Diagnosis correct on clinical grounds

When the clinical presentation is typical, some common conditions can be diagnosed on sight. The frequency and accuracy of 'spot' diagnosis will depend on the training, experience and clinical acumen of the doctor concerned.

Some examples are:

- Congenital (present at birth) – Mongolian spot, congenital naevi, birthmarks;
- Infancy – miliaria, cradle cap, napkin rash;
- Childhood infections – warts, molluscum contagiosum, impetigo, cold sores (herpes simplex);
- Exanthema – measles, rubella, chickenpox and hand, foot and mouth disease;
- Children and adolescents – acne, tattoos, melanocytic naevi, effects of trauma, fauna, flora, other environmental effects (e.g. dry skin, bush feet);
- Common genetic disorders – keloid and hypertrophic scars, dermatosis papulosa nigra, folded skin of the forehead, androgenetic alopecia (male pattern baldness) and pseudo-acanthosis nigricans;
- Cultural and traditional practices (e.g. use of ochres or chewing tobacco, lateral malleolar bursitis);
- Miscellaneous – callosities/calluses, seborrhoeic keratosis, stucco keratosis, hives (urticaria), vitiligo and xanthelasma.

However, investigations may be required to determine the cause; for example, impetigo – *Streptococcus pyogenes* or *Staphylococcus aureus*, for possible associations/causes as in hives or neuropathic ulcer, or to exclude an important differential diagnosis.

2.2 Diagnosis probable: simple confirmation needed

Conditions frequently suggested by the clinical appearances, but by no means always, are: ringworm (tinea), thrush (candidosis), pityriasis versicolor, scabies and lice (pediculosis) of the head and pubic region. The diagnosis should be confirmed by appropriate simple investigations.

Disease (cause)	Simple investigations
Ringworm/tinea (Dermatophytic fungi)	1 Wood's light – of scalp – may be positive (*Microsporum canis*) 2 Direct microscopy (potassium hydroxide [KOH] 10%) hyphae (segmented) 3 Culture to identify genus/species
Thrush/candidosis (*Candida* species)	1 Direct microscopy – yeast, some budding, pseudo-hyphae 2 Culture – for species
Pityriasis versicolor (*Pityrosporon orbiculare/ Malassezia furfur*)	1 Direct microscopy – clustered yeast, pseudo-hyphae 'spaghetti and meatballs' or 'grapes and vine' pattern 2 Culture – not usually necessary but possible
Scabies (*Sarcoptes scabiei*)	1 Macroscopic mite from burrow on needle point. 2 Direct microscopy – mite, ova, faecal pellets
Head lice/pediculosis capitis (*Pediculus humanus capitis*)	1 Direct microscopy – nits on hairs, lice (elongated body)
Crabs/pubic lice (*Phthirus pubis*)	1 Direct microscopy – lice (body round-ovoid, up to 2.0 mm diameter, crab-like)
Trichomycosis axillaris (*Corynebacterium sp.*)	1 Direct microscopy – hairs in KOH 10% 2 Gram stain – Gram-positive rods in concretions 3 Culture not necessary but can be done
Erythrasma (*C. minutissimum*)	1 Wood's light examination – coral pink fluorescence 2 Gram stain of skin scraping/tape stripping – Gram-positive rods or filaments 3 Culture – not necessary but can be done

Generally, whether a diagnosis was made on clinical grounds alone or was supported by simple confirmatory evidence, a more detailed history and a full examination of the skin should be undertaken. There are exceptions when these steps may not be necessary such as warts, male-pattern baldness, skin tags and dermatosis papulosa nigra, which may concern some people.

? 3 Diagnosis possible: further investigations required

In the 'diagnosis possible' group more detailed information will usually be needed. This may require further investigations.

Examples of conditions that may fall in this diagnostic category are common dermatoses with atypical forms or presentations; drug eruptions; contact dermatitis (primary irritant or allergic); light sensitivity; dermatitis artefacta; some systemic infections such as leprosy and syphilis; metabolic disorders, for example haemochromatosis, porphyrias; and endocrine and nutritional disorders.

In such diagnostic circumstances a detailed history, full dermatological and general medical examination, together with appropriate investigations (may include skin biopsy, blood tests) will usually be needed. Again, the solution will largely depend on the clinician concerned.

? 4 Diagnosis not possible

If the diagnosis is not possible at this stage the condition could be outside the experience of the clinician. Examples of conditions that may fall into this category for a number of practitioners are discoid lupus erythematosus (DLE); annular erythemas, atypical erythema multiforme; some presentations of granuloma annulare; necrobiosis lipoidica (diabeticorum); vesicular and bullous diseases such as dermatitis herpetiformis, bullous pemphigoid and pemphigus; various exotic diseases such as cutaneous leishmaniasis and lupus vulgaris (cutaneous tuberculosis); and the enormous range of rare conditions.

If a diagnosis is not possible, take an appropriate history and make a detailed examination then reconsider the diagnosis.

3 Take an appropriate history (see page 14)

4 Do a detailed examination of the skin (see page 16)

If the diagnosis is still not apparent, possible further management options are:

5 More invasive investigations

remembering the risk of keloid after skin biopsy in dark-skinned patients

6 Consider a trial of simple treatment

7 Request another opinion

TAKING A HISTORY IN DERMATOLOGY

Taking a good history is dependent on the communication skills of the health professional, the presence of rapport and the development of trust. Health professionals who are not part of the local service, are new or are providing a visiting service should conduct sessions with the advice of local health professionals. In particular, advice from local Aboriginal community controlled health services should be sought or clinics conducted within these services to build on the trust that has already been developed.

Aboriginal health workers are able to act as cultural brokers for difficult aspects of a consultation and should be considered essential to any consultation where communication may be a problem.

To obtain the essential features of the origin, course and development of a skin disorder, the following questions should be asked:

Onset

- Duration – days, weeks, months, years
- Site first affected – where did it first begin?
- Appearance at onset – what did it look like at first? Was it red or some other colour; smooth or lumpy; blisters or sores; did it weep, crust, bleed or show pus?

Symptoms

- Does this skin trouble cause itch, pain, heat, burning, numbness, other sensory changes such as paraesthesia (tingling, pins and needles), formication (crawling or creeping sensation), appreciation of heat and cold, alterations in sweating, other feelings?

Course

- Has the trouble stayed much the same, got worse or got partly or completely better?
- If the trouble has spread, in what order and when were the different parts of the body affected?
- What changes in appearance have there been?
- Rate, regularity, manner of change
- Remissions and relapses and their respective time sequences
- What effects have treatments had?

Apparent predisposing and precipitating causes

- What seems to have brought it on?

Aggravating and relieving influences

- What makes it better or worse – time of day, climate, season, sunlight, heat or cold, work, food, alcohol, menses, baths and showers, getting hot and sweaty, being upset, etc.? Has any treatment made it better or worse?

Patient's ideas about the condition

- What do you think is the problem?

Previous attacks and treatment

- Has this happened before? When? What helped last time?

Others affected in the family or in the environment

- Friends, fellow workers, other members of the local community

General health

- Other symptoms currently or recently – fever, joint pains, sore throat, headache, malaise
- Past medical history
- General family history – Skin problems
  – General (e.g. diabetes mellitus, hereditary diseases)
- Allergies to medications

Personal history

- Occupation, recreations, use of alcohol, tobacco, other drugs

Five questions:

If the eruption looks odd or bizarre, does not conform to the common skin diseases, or a diagnosis cannot be suspected or made on clinical grounds, five questions should be asked again as a routine.

1. What drugs or medicines (prescribed, over-the-counter preparations, traditional, and given by relatives and friends) are/have been taken internally for this or other conditions – including analgesics (pain killers), sedatives (sleeping tablets), tranquillisers, laxatives (bowel-opening medicines), contraceptive pills?
2. What local applications have been used – prescribed, traditional or over-the-counter from pharmacies, supermarkets, health shops, doctors, nurses, relatives, friends?
3. Is the patient sensitive or allergic to any drugs/preparations used internally or externally?
4. What are the patient's present and previous occupations, hobbies and recreations?
5. Where and when has the patient travelled and lived?

CLINICAL EXAMINATION IN DERMATOLOGY

Clinical examination of the skin is easy – given a keen observer, a good routine and some simple equipment. In addition, the clinician must know what he/she is looking at – and looking for – in a particular patient. Clinical examination of the skin should present no difficulties if a well-tried system is followed, a few simple aids are used and the examiner has a knowledge of skin lesions and their significance.

Use daylight rather than artificial light

Patients with skin changes should be examined in as much daylight as possible. Window blinds should be pulled right up instead of being pulled partly or fully down. The examiner should be between the source of light and the patient. The examiner should not look into the light and should not cast a shadow on the surface being examined.

Artificial light may have to be used to examine areas such as the mouth and the genitalia. A source of artificial light must give sufficient illumination and the nearer this approximates daylight the better. Macules and some other changes in skin colour are not easily seen in artificial light, especially in patients with dark skin.

Despite the need for proper light, the patient's privacy and modesty must be respected by using suitable screens appropriately placed before, during and after the examination. When patients of the gender opposite to the examiner are being seen, a chaperone, preferably of the same gender as the patient, should be present during the interview and examination.

Examine the entire skin surface

A good rule is to examine the entire body surface. Patients sometimes deny the presence of lesions other than the skin surfaces they are prepared to offer for examination. At other times, patients have significant lesions they have never seen and, for this reason, will also deny their presence. The need to examine the entire skin surface can make difficulties and cause embarrassment for both patient and examiner regardless of the patient's gender or age; hence, examiners should be thoughtful, tactful and gentle and explain why such a detailed examination is necessary.

Although examination of the entire body surface is time consuming and is resisted by some patients, mistakes will be made and conditions missed if a complete and thorough examination of the skin, its appendages and mucous membranes is not done.

Hair, nails and mucous membranes need examination

The hair, nails of the fingers and toes, and the mucous membranes of the mouth and throat, and often of other areas such as the eye, vulva, vagina, urethra and rectum, may need to be included in the examination of some dermatological patients.

The hair includes that of the scalp, eyebrows, eyelashes, beard, axillae (armpits), pubic region and body.

Dermatological examination is more than visual

Examination of the skin should not be restricted to visual inspection.

Palpation, that method of examination by touching parts of the body using the hands, is often essential in examining the skin. Certain cutaneous changes including tenderness, induration, oedema and altered elasticity can be best evaluated if the examiner touches the patient's skin.

When the condition is not contagious, the attending doctor should deliberately touch the patient's skin during the examination. This gesture, supported by a reassuring explanation about the condition, will usually allay the anxiety and reduce the fears of contagion so often felt by patients, their relatives and their associates.

Grattinage is a combination of light scraping and scratching aimed at breaking up scale to show the features and to reveal the underlying surface. A piece of clear perspex such as a ruler can be used, but the examiner's index fingernail is usually more readily and conveniently available and it allows a better appreciation of the texture, quality and other features of the scale.

Grattinage is useful, for example, in pityriasis versicolor and ringworm, both common in many northern Australian Aboriginal communities. The scale in pityriasis versicolor is usually easily and quickly removed. In ringworm of the body (tinea corporis) due to *Trichophyton rubrum* (granular variant), scale is usually removed with great difficulty, often to the point of superficial bleeding when using a scalpel blade.

Measurement is as important in dermatology as in other branches of medicine. The size of lesions is important in records of clinical findings, in assessing progress and determining treatment. The size of lesions should be measured and recorded in centimetres and millimetres and not as, say, the size of a fingernail or other common objects. A ruler made of clear perspex and graduated in centimetres and millimetres is both cheap and continually useful.

Pressure on lesions is often useful in dermatological diagnosis. This technique is called **diascopy**. It is defined as the use of a transparent object,

such as a piece of clear perspex (e.g. ruler), a glass slide or drinking glass, to see whether the lesions fade, change colour or decrease in size when steady and even pressure is applied for at least 10 seconds.

Glass slides should be used in diascopy with care in case they break and cause injury.

Diascopic examination allows distinction to be made between erythema and telangiectases (in which blood is contained within the blood vessels) and purpura and ecchymoses (in which the blood is outside the blood vessels); the former two disappear under pressure; the latter do not.

Demonstration of **alteration in sweating and sensation** is of great diagnostic importance in leprosy and other neuropathic diseases. The first can be shown clinically by painting tincture of iodine, allowing it to dry, exposing the painted area to a source of heat or exercising the patient, and then applying a light dusting of starch powder over the painted surface. The combination of iodine, sweat if present and starch will produce a blue colour.

A pin, a wisp of cotton wool, string or soft tissue paper and a test tube of hot or cold water will allow a clinical assessment of altered sensation.

Changes of skin temperature that occur in peripheral vascular disease (decreased, cool) or inflammation such as cellulitis (increased, warm), can often be judged clinically by simple palpation, preferably using the backs of the fingers.

Magnification of the skin can be revealing

No matter whether the examiner's eyes are young and untrained or old and experienced, magnification will prove invaluable and indispensable. The use of a magnifying glass or hand lens (x2–10), binocular loupe or small hand-held epiluminescence microscope (Episcope, Dermatoscope) will show structural details of skin lesions more clearly and in greater detail than with the naked eye.

The pseudocysts of a seborrhoeic keratosis or uniform redness of vascular lesions can be clearly distinguished from benign and malignant melanocytic lesions.

General medical examination

The skin often shows signs indicative of disease in other organs and systems of the body. General medical examination and assessment is therefore needed to reach a correct diagnosis.

Sometimes enlargement of the lymph nodes in the neck is the first sign of head lice. Recurrent skin infections and vulval itch (pruritus) are sometimes associated with diabetes. Internal malignancy may first present with clinical signs in the skin.

The associations between the skin and systemic diseases are virtually endless and should always be remembered when dealing with patients with dermatological conditions.

Examinations beyond the skin

A visit to where the patient lives or works to see and assess the conditions in which the complaint developed can sometimes be most helpful. Health professionals have a responsibility to be advocates for improvement in the living conditions of many Aboriginal people. An awareness of the high prevalence of common skin conditions such as fungal and bacterial infections as a result of poor socio-economic circumstances can help to galvanise action from within the profession for uptake of policy to address these health determinants.

Before these procedures are carried out, the matter must be discussed in simple, clear words. The method, main instruments to be used and the reasons for the procedure should be explained by the doctor to the patient and their agreement obtained in writing if possible.

Where specimens are being sent to a laboratory, be sure the specimen is accurately labelled with patient details, site and type of specimen. The request slip for the pathologist needs as much clinical detail as possible.

The site of collection for each specimen, the type, the date, person's name, age, gender and place of living should be recorded on each package, in the operator's record and the procedure record of the patient's medical card.

Wood's light examination

Wood's light is ultraviolet from which the visible rays have been excluded by Wood's glass or filter. The filter mainly consists of barium silicate containing about 9% nickel oxide. It transmits rays of wavelengths around 345–365 nm.

Indications

Wood's light can be used as an aid in the diagnosis of:

1 *Microsporum canis* (*M. canis*) tinea capitis (scalp ringworm). The infected scalp hairs fluoresce a greenish-yellow similar to an illuminated watchface in the dark.
2 Pityriasis versicolor fluoresces on the body as a greenish-yellow.
3 Alterations in pigmentation can be more obvious or less apparent (e.g. more obvious in pityriasis versicolor and vitiligo but not in leprosy).
4 Porphyrins fluoresce coral pink. They are produced by *Corynebacterium* species on the skin and in the pores of the nose. Erythrasma, a corynebacterial infection of the flexures, will fluoresce coral pink. Maceration between the toes secondarily infected with corynebacteria will also fluoresce. The normal pink fluorescence seen in the pores of the nose will be absent in a patient taking antibiotics. The porphyrins produced by corynebacteria are water soluble and will wash off.

Limitations

The limitations and causes of mistakes include:

1 Ointments, mineral oils, some deodorants, make-up, soaps and some contact sensitisers may fluoresce a blue or purple colour.
2 Reflection from white clothing.
3 Optical brighteners in washing powders may stick to hair and fluoresce as pink speckles.

- Of the dermatophytes found in Australia, only *M. canis* infecting scalp hairs will fluoresce. *Microsporum canis* infection of other sites does not fluoresce.

Method

Wood's light must be used in a completely darkened room. Before use, the operator should allow sufficient time for vision to become adapted to the dark and the Wood's light turned on for 3–4 minutes before use to allow the unit to warm up. The light should not be directed into the patient's eyes. The skin should be well-cleaned before the examination, except when trying to detect porphyrins. The observer should stand behind the seated patient and hold the light unit in the hand about 10 cm from the patient's scalp or skin. When the face or beard area is examined, the patient's eyes should be closed and shielded with opaque material.

Specimen collection for fungal (dermatophyte) microscopy and culture

As a general rule, the greater the amount of skin, hair and nail taken, the greater are the chances of positive results in patients with ringworm.

Indications

- For suspected tinea/ringworm or yeast infections of skin, hair or nails.

The aim of direct microscopy on suitably prepared wet mounts of specimens of skin, hair and nails is to find and identify hyphae and spores. Their presence is highly suggestive of a dermatophyte. However, failure to find fungal elements on direct microscopy does not prove the absence of a dermatophyte.

Precautions

Dermatophytes in skin, nails and hair will remain viable for about 30 days – perhaps longer. However the longer the delay between collection of specimens and processing, the greater is the possible deterioration of specimens and likelihood of contamination. Should it seem the 30 day period may be exceeded because of transport difficulties, culture media are best inoculated at the time when specimens are taken. A selective Sabouraud agar (SSA) in McCartney bottles (3 cm diameter) and small Petri dishes (6.5 cm diameter) are suitable for field work in remote areas. Culture media should be kept in a refrigerator (not in the freezer section) and carried in an Esky with a frozen ice brick. This medium will keep satisfactorily for 2–3 months under these conditions.

If scraping skin and nails indoors, overhead fans must be stopped and sources of draughts such as open doors and windows must be closed. If outdoors, collection of skin and nail specimens can be difficult or impossible on windy days so that some sort of shelter becomes obligatory.

Two points should be emphasised:

1 The appearance of the hyphae and arthrospores in skin specimens is not indicative of the genus and species of the dermatophyte.
2 *Candida albicans* is a yeast which can produce pseudo-hyphae that can be confused with the hyphae of dermatophytes.

Methods

1 Skin Scrapings

Cleaning the skin

When practical, patients should be told not to use any local applications such as ointments, creams and lotions for at least two days before specimen collection. In field work this is not usually possible. Any residual local application should be removed using soap and water, alcohol spirit or acetone. If the area of skin selected for scraping looks reasonably free of dust, dirt, local applications and other accumulations, cleaning is not necessary. If the skin is cleaned, it should be dried before taking the scraping.

Sites to scrape

Promising areas to select for scraping are any raised, spreading edges of the rash, especially if recently developed, and vesicular borders. In these places, hyphae can be expected to be numerous. Macerated skin in interdigital sites is not satisfactory for examination. Scrapings should be taken from the affected part bordering normal skin.

Instruments

Sterile single-use scalpel blades in individual foil packs are generally favoured. These are particularly sharp and careful use is essential to avoid cutting both patient and collector. Used blades must be disposed of safely. One-piece scalpels, tweezers and curettes are blunt and therefore safer to use for infants, young children and in awkward sites such as between the toes. They have the practical disadvantage of the need to clean and sterilise each time after use.

Collection of scrapings

Skin scrapings can be conveniently collected on a piece of paper about 15 x 10 cm (e.g. from a notepad). The sheet can be folded sharply to make a gutter held firmly against the skin by the patient or nurse or with adhesive tape.

Scraping the skin

The skin near the selected site should be stretched in one direction with the operator's thumb and index finger of one hand and the chosen instrument firmly scraped in the opposite direction, across and away from the spreading edge if one is present. The scalpel blade should be held obliquely to the skin surface. It is remarkable how firmly the skin must be scraped to obtain sufficient material for direct microscopy and culture. At times, superficial bleeding results. These comments apply particularly to *Trichophyton rubrum* (granular variant). Should bleeding occur, the site should be covered and the patient reassured. A rough guide to a minimum quantity of skin required is the heads of two burnt safety matches crushed to reasonably fine particles. Tops of vesicles can be removed with a scalpel blade. Skin scrapings should be taken from several areas in one site. If more than one site is affected, separate specimens should be taken using a new scalpel blade or sterile instrument for each site. Where the horny layer is thick, as on the palms and soles, deep scraping is needed.

Collection of Hairs

Broken hairs near the advancing border should be pulled out with tweezers flattened along the gripping edge. Those sold in chain stores and supermarkets and used to pull out eyebrows, nasal hairs and splinters are cheap and unsatisfactory. If a Wood's lamp can be properly used, individual hairs that fluoresce can be selected for direct microscopy, culture and other investigations. Hairs that are broken and lustreless should be pulled out so the ends extend beyond the gripping edge of the tweezers. Infected hairs often show a white opacity around the root. Normal hairs are more translucent. Hairs infected by endothrix infection (e.g. *T. tonsurans* and *T. violaceum*) are so often packed with spores and digested by the fungus that they are twisted and broken when they reach the surface of the scalp. Then the tweezers should be firmly pressed into any scale present before scraping or plucking in attempts to collect pieces of broken infected hairs. Affected areas of the scalp should not be scraped with a sharp scalpel or blade as this cuts or breaks infected hairs above the part invaded by the fungus. Removal of scale with a blunt scalpel or with tweezers often reveals broken hairs.

Brush samples of scalp scale and hairs uses round plastic brushes 8 cm in diameter passed firmly over the scalp approximately ten times and then lightly pressed into 9.0 cm Petri dishes containing the appropriate medium. The brushes can be re-used after washing thoroughly with soap and water then soaked in disinfectant.

When ringworm presents as kerion (severe inflammatory tinea), specimens are usually difficult to obtain as hairs are shed as a result of the acute inflammatory process. At times broken hairs can be found and pulled out from the edge. Specimens of pus should be taken for bacteriological studies. While kerion can result from fungus infection, secondary bacterial infection does occur and should be assessed.

3 Nail Clippings

Specimens can be difficult to obtain especially when the nail is thickened, hard and dystrophic. The nail should be clipped, scraped and pared until the crumbling degenerate part is reached. Debris underneath the nail should also be collected.

Packaging specimens for dispatch

When sufficient skin, nail or hair to be easily seen has been collected, the scraping edge of the instrument can be carefully wiped on one side of the collecting gutter. By light tapping, the skin/nail scrapings or collected hairs can be centred in the gutter, obvious hairs removed if not required, the paper folded several times, the ends turned down and the resulting small packet sealed with adhesive tape. Specimens can also be carried between two glass slides, the ends and sides being sealed with adhesive tape and labelled with patient details. A cardboard slide carrier to reduce the risk of damage in transit is advisable. Specimens may also be collected directly into or transferred for dispatch in clean plastic specimen bottles. Specimens can also be inoculated directly onto culture media as described earlier.

Bacterial swab for microscopy and culture

Indications

- For bacterial infections such as streptococci, staphylococci.

Not suitable for atypical bacteria such as mycobacteria.

Method

1 Using sterile swab, collect pus or other discharge or swab the base of the lesion for immediate insertion into sterile transport medium for culture.
2 The swab may need to be moistened in sterile normal saline if the lesion is dry and not discharging.
3 Using a second swab, collect further material to smear directly onto glass microscope slide for Gram stain and microscopy.
4 Appropriate culture plates may be inoculated directly.

Skin scraping for scabies microscopy

Indications

- For suspected scabies including crusted (Norwegian) form.

Method

- Scrapings should be taken from possible burrows, most commonly in the fingerweb spaces, but also may be found around the wrists, elbows, axillary folds, nipples, penis and feet.
- In infants scrapings from palms, soles and scalp can also be taken.
- For crusted scabies scrapings can be taken from any scaly site.
- Using a #15 scalpel blade scrape along the length of the burrow, with the blade parallel to the long axis of the burrow.
- Spread onto glass microscope slide.
- Adult mites can sometimes be extracted from the vesicle at the end of the burrow using a fine needle.
- Potassium hydroxide (KOH) will dissolve keratin, which may obscure the mite, faecal pellets and eggs (ova). This will occur more quickly with the addition of dimethyl sulphoxide (DMSO).
- The adult mite is just visible to the naked eye and therefore is easily seen by low power of the microscope.
- More commonly, however, the eggs and faecal pellets will be found by a higher power and more careful examination of the slide.

Tzanck smear

Indications

- For cytopathic effect of herpes viruses – varicella zoster (HVZ) and simplex (HSV).

Can also show acantholytic cells (e.g. pemphigus), bacteria (e.g. bullous impetigo).

Method

Scrape floor of blister with scalpel blade and smear onto glass slide. Air dry or fix in absolute alcohol. Stain (e.g. haematoxylin and eosin) and look for multinucleate giant cells in herpes virus infections (i.e. uniform rounded cells with a large nucleus in pemphigus).

Viral immunofluorescence studies

Indications

- For herpes viruses – HSV types 1 and 2 (cold sores, genital herpes), HVZ (chickenpox/shingles).

Method

Scrape floor of blister/vesicle with scalpel blade, smear onto glass microscope slide and fix in acetone. The laboratory can perform immunofluorescence for HVZ, HSV-1 and HSV-2.

Dermal smear for lepromatous leprosy

Adapted from JC Hargrave and ER Jones. *Leprosy in Tropical Australia: A Manual for Field Workers*. Northern Territory Medical Service, Darwin, 1980.

1 Wash a new microscope slide thoroughly in soap and water, rinse it in methylated spirit and dry it with a clean towel.
2 Clean the earlobe, forehead or lesion from which the smear is to be taken and let it dry.
3 Gently squeeze the earlobe or lesion until it becomes bloodless.
4 Make an incision about 0.5 cm long and 1.0 mm deep with a new scalpel blade and immediately scrape the edge firmly. The tissue fluid obtained should then be spread on the microscope slide.
5 'Fix' the smear by passing the underside fairly quickly over a naked flame a few times. (A naked flame could be a propylene cigarette lighter or primus stove.)

The smear will be examined for *Mycobacterium leprae*, acid-fast bacilli.

Skin biopsy

Indications

May be required to make or confirm a diagnosis, particularly of an inflammatory or neoplastic condition and some infections (e.g. deep fungi, atypical mycobacteria).

- For histology, immunofluorescence, culture.

Precautions and other considerations

Will leave a scar that carries a high risk of becoming hypertrophic or keloid in dark-skinned patients.

Antibiotic prophylaxis – consider if there is a history of infective endocarditis, rheumatic heart valve disease, mitral valve prolapse or heart valve surgery/replacement.

Ask about any treatment in the past 48 hours, as this may influence the histological appearance.

Ask about reactions to injections, local anaesthetics or dressings.

Select the biopsy site carefully – this will be influenced by the site or distribution of the lesion/eruption, age of the lesion(s), differential diagnoses,

underlying structures that may be damaged by the biopsy (e.g. temporal branch of facial nerve is close to the surface when crossing the temple) and the risk of keloid.

Equipment required

- Specimen container(s)
- Disposable dressing pack with gauze, cotton balls.
- Alcohol swab/skin antiseptic such as aqueous chlorhexidine.
- Disposable sterile needle/syringe – e.g. for the face, digits a ultrafine 1 mL (100 U) diabetic needle/syringe in a single unit is recommended; elsewhere recommend luer-lock to avoid the needle and syringe disconnecting and spraying local anaesthetic into your face!
- Disposable sterile punch biopsy. These come in a range of diameters from 2–8 mm. Most commonly use a 4 mm or 3 mm – avoids the need to clean and sterilise, difficult inside the barrel.
- Single-use vials of local anaesthetic – most commonly 1% lignocaine with 1 : 100 000 adrenaline or plain 1% lignocaine.
- Sterile fine-toothed forceps or skin hook to grip the specimen.
- Sterile fine scissors or scalpel blade to cut the deep aspect of the specimen.
- May require suture/needle holder – depending on site and size of biopsy and likelihood of removal of sutures later.
- Dressings.

Method for a punch biopsy

Lie the patient down.

Clean biopsy site with an alcohol swab/antiseptic.

Infiltrate with local anaesthetic – usually 1% lignocaine with 1 : 100 000 adrenaline; maximum lignocaine dose should be 7 mg/kg; avoid using adrenaline at end-organ sites where arterial contraction may compromise tissue viability (e.g. digits, penis, ear).

Wait for the effects – the local anaesthetic effect on reducing pain is usually rapid in onset (within 2-3 minutes, maybe longer); however, it takes longer to numb light touch. The adrenaline effect on vessel constriction takes 15–30 minutes.

Label the specimen container(s). Recommend 10% buffered formalin for routine histology; a little normal saline for fresh specimen taken for immunofluorescence if able to transport to laboratory immediately, or Michel's transport medium if there will be any delay (within 24 hours); sterile normal saline and gauze in a sterile container for culture.

Complete details on pathology request slip including as much clinical detail as possible (site of biopsy, distribution of eruption, duration, clinical description, treatments tried) and a list of differential diagnoses. List tests requested (e.g. histology, special stains, immunofluorescence, culture for what organisms).

Check site is numb.

Immobilise the biopsy site between the thumb and index finger of the non-dominant hand.

Hold punch biopsy perpendicular to the skin and press firmly against the skin. Then with a twisting, rotating back and forward motion push the punch into the skin to cut a core of skin. Aim to penetrate to the hilt of the metal part of the instrument, which for most sites will reach the subcutaneous fat. Withdraw the instrument.

Pick up the skin core with the toothed forceps or skin hook. Consider where the likely pathological process is occurring so that the important part is not squashed. Generally try to pick up as deep as possible in the subcutaneous fat layer.

Promptly cut the core free at the deepest possible level using fine scissors or scalpel blade.

Transfer to the specimen container, taking care to put it in the correctly labelled one if more than one biopsy is being taken.

Haemostasis – may include simple pressure, haemostatic applications, diathermy and/or suture.

Suture if required – remember the risk of hypertrophic and keloid scarring from the suture holes.

Dressing – the biopsy site should be covered with pressure to reduce the risk of bleeding and with occlusion for the first 24 hours to promote healing.

Wound care

The dressing should be changed daily after cleaning the wound with an antiseptic such as povidone-iodine until healed (1–2 weeks).

More frequent dressing changes may be required if there has been considerable bleeding or oozing.

Deep incisional biopsy

Deep incisional biopsy is required when the pathological process may involve the subcutaneous fat (e.g. panniculitis). A punch biopsy is not suitable as it does not collect sufficient subcutaneous fat. Using a #15 scalpel blade take a deep ellipse (approximately 2–3 cm long) into the subcutaneous fat; will require suturing.

Section 2

Selected Conditions in Detail

Folded Skin of the Forehead

Synonyms: Gyrate skin, cutis gyrata

Summary: Persistent and readily visible folds of skin on the forehead. More likely to be noticeable in adult males. Is of no clinical significance but may be mistaken for leonine facies of lepromatous leprosy.

CLINICAL DESCRIPTION

- Permanent deep folds of the forehead;
- Bilateral and symmetrical;
- Can be progressive.

Early

- Two vertical linear depressions between the eyebrows above the root of the nose (glabella);
- Skin between the folds appears smooth and increasingly prominent, then horizontal folding of the forehead initially central, extending towards the temples.

Late

- Also vertical folding of the frontal crown;
- May also involve the temporo-parietal scalp, running from front to back.

Epidemiology

- Onset from the late 20s;
- More common in males than females.

Causes

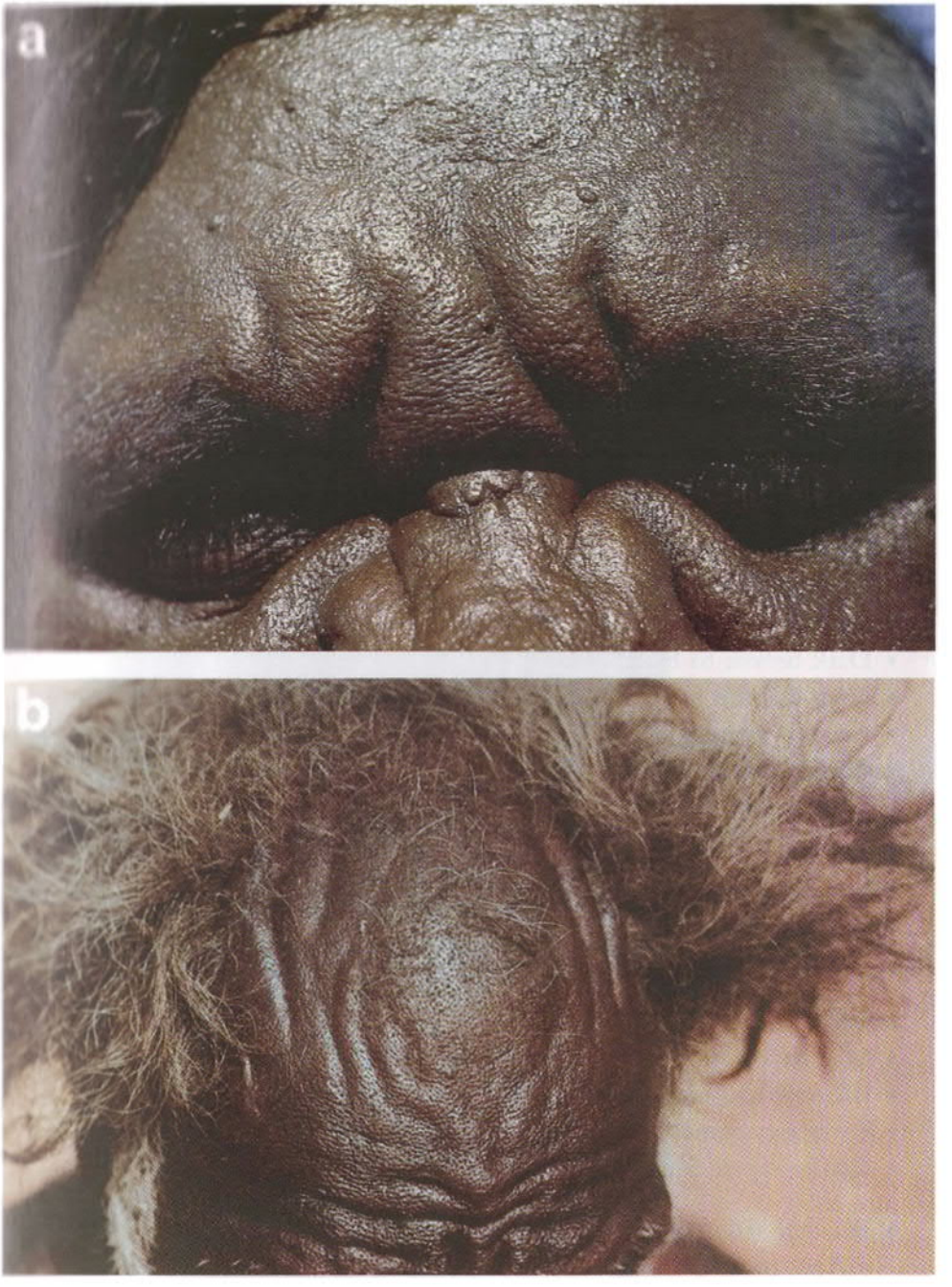
- Genetic;
- Hypermobility (?) of forehead muscles – corrugators and frontalis leading to hyperkinetic facial lines.

Making the diagnosis

- To distinguish from the leonine facies of lepromatous leprosy:
  - Clinical history/examination for other features of lepromatous leprosy
  - Investigations (see pages 96-99).

Significance

- Is of no clinical significance but may be mistaken for leonine facies of leprosy.



a *Early stage just involving the glabella and root of nose.*
b *Late stage involving the forehead, temples and frontal crown of scalp.*

Dermatosis Papulosa Nigra

Summary: Benign skin condition that is highly prevalent in dark-skinned people, not unlike seborrhoeic keratoses. Is of no clinical significance except for cosmetic reasons or in excluding a diagnosis of unrelated conditions.

CLINICAL DESCRIPTION

- Site:
  - Usually occurs around the eyes; that is, over the cheekbones and temples;
  - May extend more widely onto the forehead, eyelids and sides of neck down to the collarbones (clavicles);
  - Rarely involve the nose, lips, ears and scalp.
- Discrete, hemispherical small raised spots (papules);
  - Some develop a stalk (pedicle).
- Round or oval, sometimes polygonal in shape;
- Dark brown to black colour;
- Firm but not hard;
- Are not grouped and do not occur in lines;
- Asymptomatic (not itchy or painful); but those on stalks may become annoying, especially on eyelids.

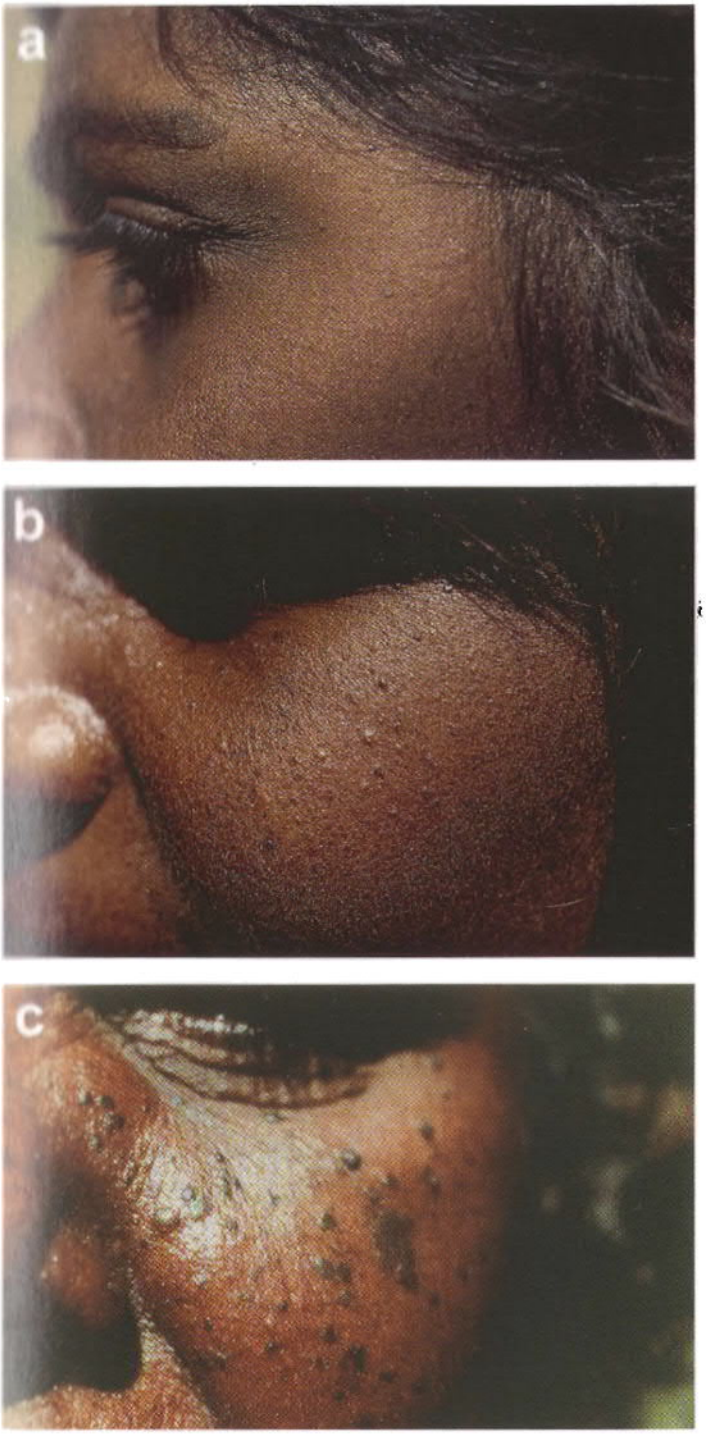
Early

- May be solitary or just a small number; asymmetric distribution;
- Smooth surface;
- Small (< 1 mm in diameter).

Late

- May become very numerous – up to 150 or more;
- Bilateral and fairly symmetrical – same on both sides;
- Surface becomes warty (verrucose);
- Can be up to 5 mm in diameter.

- Become larger and more prominent with age;
  - Appearance of new papules is generally slow over a number of years;
  - May progress rapidly in some post-menopausal women;
- Individual papules seem to persist indefinitely;
- Cancerous change does not occur;
- Systemic changes or associations have not been reported.



a *Early development.*
b *Mid-development.*
c *Later development of dermatosis papulosa nigra.*

Epidemiology

- Common in most dark-skinned groups;
- Onset usually from early adolescence, but sometimes in late childhood or adulthood;
- Perhaps more common in females;
- Widespread throughout Australia.

Causes

- Genetic;
- Benign naevoid condition, which develops from defects in the pilosebaceous follicles;
- May be of cultural significance (e.g. part of the Dreamtime).

Making the diagnosis

- Clinical examination.
- Histology generally not required but is similar to seborrhoeic keratosis – irregular acanthosis and hyperkeratosis, increased melanin throughout the epidermis, dilated hair follicle openings forming keratin cysts, immature pilosebaceous follicles. Shave biopsy rather than punch biopsy would be sufficient and would minimise the risk of keloid scarring.
- May need to distinguish from plane warts (see page 117) or molluscum contagiosum (see page 119) (clustered and in lines), adenoma sebaceum (other features of tuberous sclerosus), melanocytic naevi (usually solitary or few in number) and skin tags.

Significance

- Benign; is of no clinical significance.

Discoid Lupus Erythematosus (DLE) of the Face

Summary: Inflammatory skin changes with alteration in colour, scaling and eventually scarring, which may be associated with systemic disease.

CLINICAL DESCRIPTION

- Butterfly distribution over cheeks is most common site on the face. May also involve the lips, forehead, scalp and upper trunk (discussed further on pages 48 and 66);
- Sharply defined;
- Circular shape.

Early lesion

- Darker than surrounding skin (pigmented);
- With some thickness that can be felt with the finger (plaques);
- Often red at the edges (peripheral erythema);
- Scaly – can be difficult to pick off;
  - Fine spicules on under-surface of the scale (tin tack sign) resulting from scale extending into the hair follicle opening (follicular plugging).

Late lesion

- Loss of pigment – white skin;
- Epidermal atrophy – thinning of skin;
- Scarring – loss of hairs.

Epidemiology

- Common in Aboriginal populations
  - prevalence estimated 1 : 1000–1500;
- Onset usually between puberty and the late forties; rarely after fifty;
- Sisters have been affected;
- More common in females than males (4 : 1);
- Widespread throughout Australia.

Cause

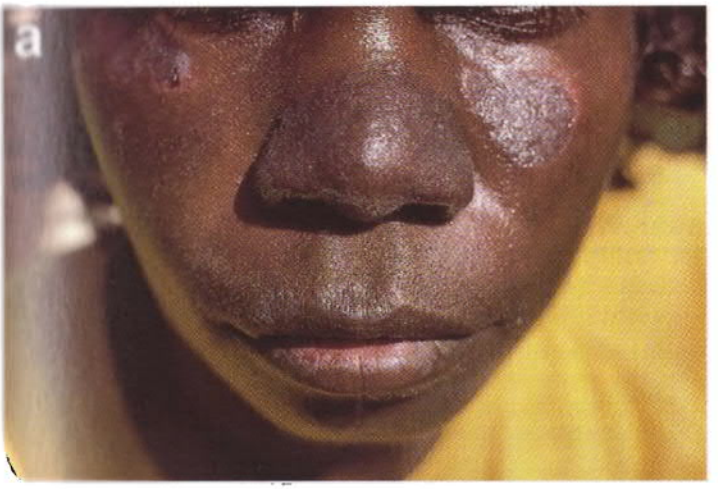
- Not known – possible predisposing factors may include genetic susceptibility, sun exposure and general health.

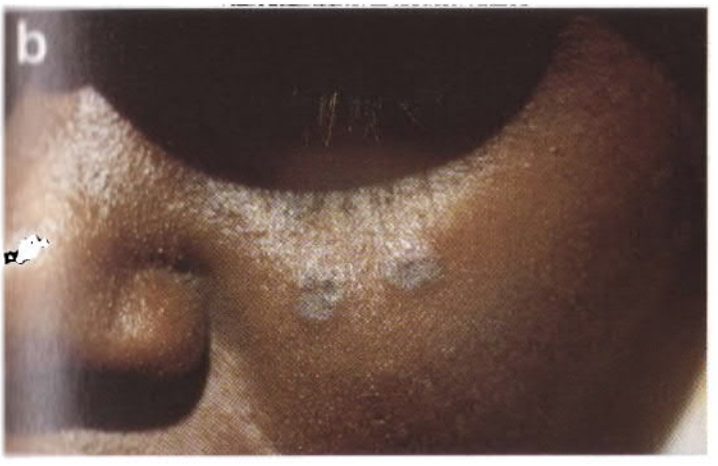
Making the diagnosis

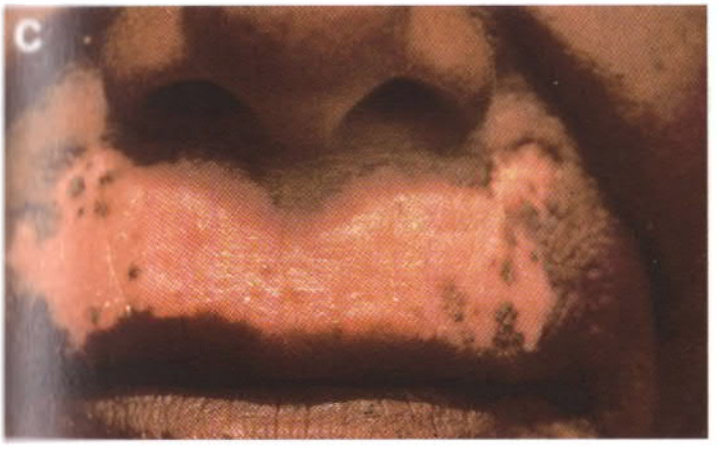
- May need to distinguish from ringworm of the face (tinea faciei) – skin scraping for fungal microscopy and culture must be performed to make this diagnosis.
- Requires skin biopsy:
  - 4 mm punch biopsies for histology and for immunofluorescence studies;
  - Histology – thinned epidermis with loss of rete ridges, hyperkeratosis and parakeratosis, follicular plugging, basal layer degeneration, perifollicular lymphocytic infiltrate;
  - Immunofluorescence – positive in 75%; often negative in early lesions of less than 8 weeks in duration; granular pattern of immunoglobulin and complement at the dermo-epidermal junction.
- Investigations for systemic lupus erythematosus are required.

Significance

- For the diagnosis of systemic lupus erythematosus and its complications;
- Squamous cell carcinoma can develop in discoid lupus erythematosus usually of the lower lip.







- *Pigmented plaques of discoid lupus erythematosus (DLE) in a butterfly distribution over the cheeks and lower lip.*
- *Small circular pigmented plaques of DLE on the cheek.*
- *Late stage DLE above the upper lip.*

Impetigo

Synonym: School sores.

Summary: Bacterial infection of skin with typical honey-coloured crusting, which may result in kidney and heart disease.

CLINICAL DESCRIPTION

- Early lesion – blister, but does not last long;
- Honey-coloured crusting;
- Satellite lesions – similar smaller spots spreading out from the original main one;
- May cause acute post-streptococcal glomerulonephritis (PSGN);
- May be linked with acute rheumatic fever (ARF) and rheumatic heart disease (RHD).

Epidemiology

- Most common in children;
- Spread by touch.

Causes

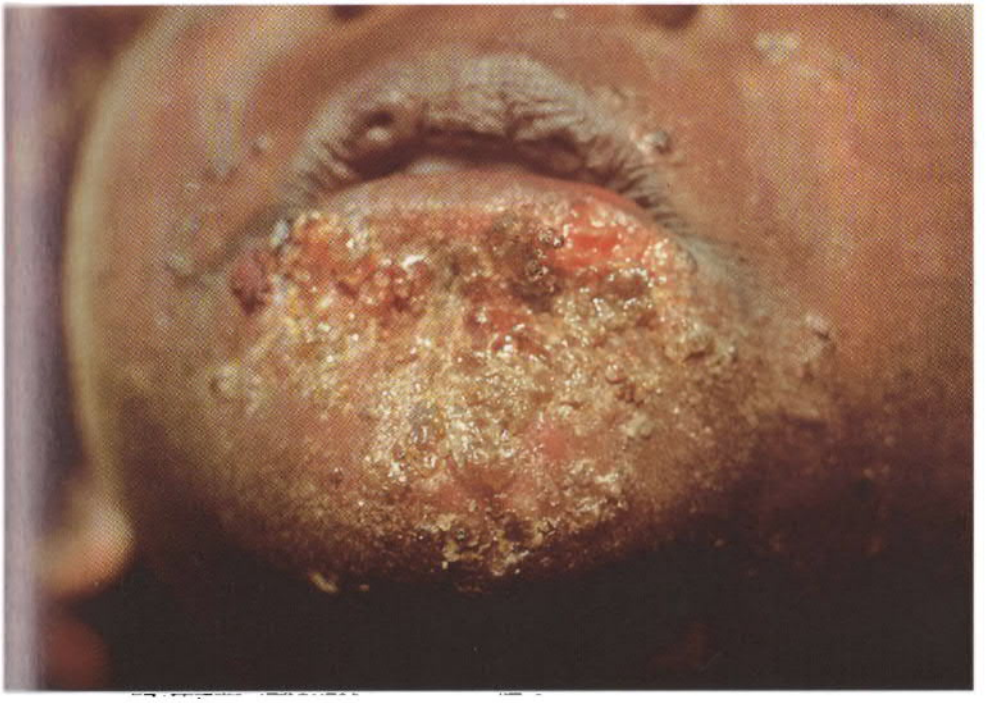
- Bacterial infection: *Streptococcus pyogenes* (Group A Strep). In Aboriginal communities, this is by far the most common cause;
  – Less commonly *Staphylococcus aureus*.
- May be primary infection;
- May be secondary to scabies, head lice, abrasions and lacerations, bites and stings.

Making the diagnosis

- Skin swab for bacteriology (microscopy and culture) including antibiotic sensitivities;
- Further investigations for PSGN and RHD.

Significance

- Risk of developing post-streptococcal glomerulonephritis and possibly RHD.



Primary impetigo on the chin.

Miliaria

Synonym: Heat or sweat rash.

Summary: Tiny clear blisters seen mainly on the face of young children and infants resulting from blocked sweat ducts in hot humid conditions.

CLINICAL DESCRIPTION

- Small clear blisters (vesicles);
- Mainly on the forehead and cheeks;
- Can involve the body.

Epidemiology

- Predominantly affects infants and young children;
- Hot humid climates may predispose the condition.

Cause

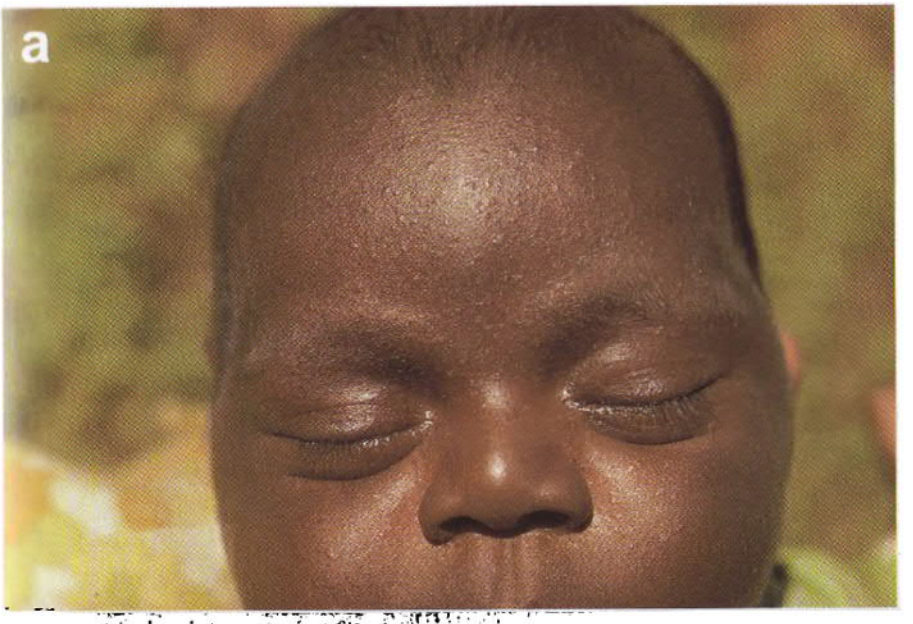
- Blocked sweat ducts.

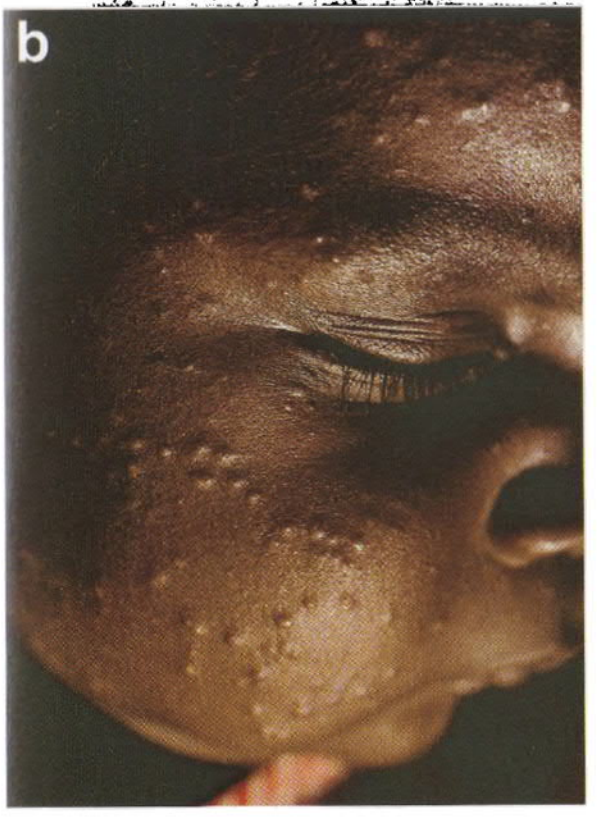
Making the diagnosis

- Usually clinical;
- May need to distinguish from chickenpox (herpes varicella zoster virus (HVZ)) or herpes simplex virus (HSV) infection by:
  - Tzanck smear;
  - Direct immunofluorescence examination.
- Trial of therapy – accommodation in an air-conditioned room can result in resolution within 12 hours;
- Skin biopsy rarely required – histology shows sweat duct obstruction with or without inflammation:
  - Miliaria crystallina – superficial obstruction;
  - Miliaria profunda – deeper obstruction.

Significance

- Is of no clinical significance.





a *Tiny clear vesicles on the forehead.*
b *Small scattered vesicles over the face.*

Acne

Synonym: Pimples, zits.

Summary: Seen most commonly on the face. In Aboriginal teenagers this is mainly non-inflammatory with blackheads and whiteheads, which may affect self-esteem.

CLINICAL DESCRIPTION

- Mainly forehead is affected but may occur anywhere on the face, trunk and upper arms;
- Predominantly comedonal – blackheads and whiteheads;
  – Skin feels rough and bumpy without inflammation;
- Papules, pustules, cysts less commonly seen;
- Cystic acne (boil-like) on the back and over the breastbone (presternal) may result in keloid scarring.

Epidemiology

- Perhaps less common than in non-Aboriginal populations;
- Most commonly seen in teenage and early adult life.

Causes

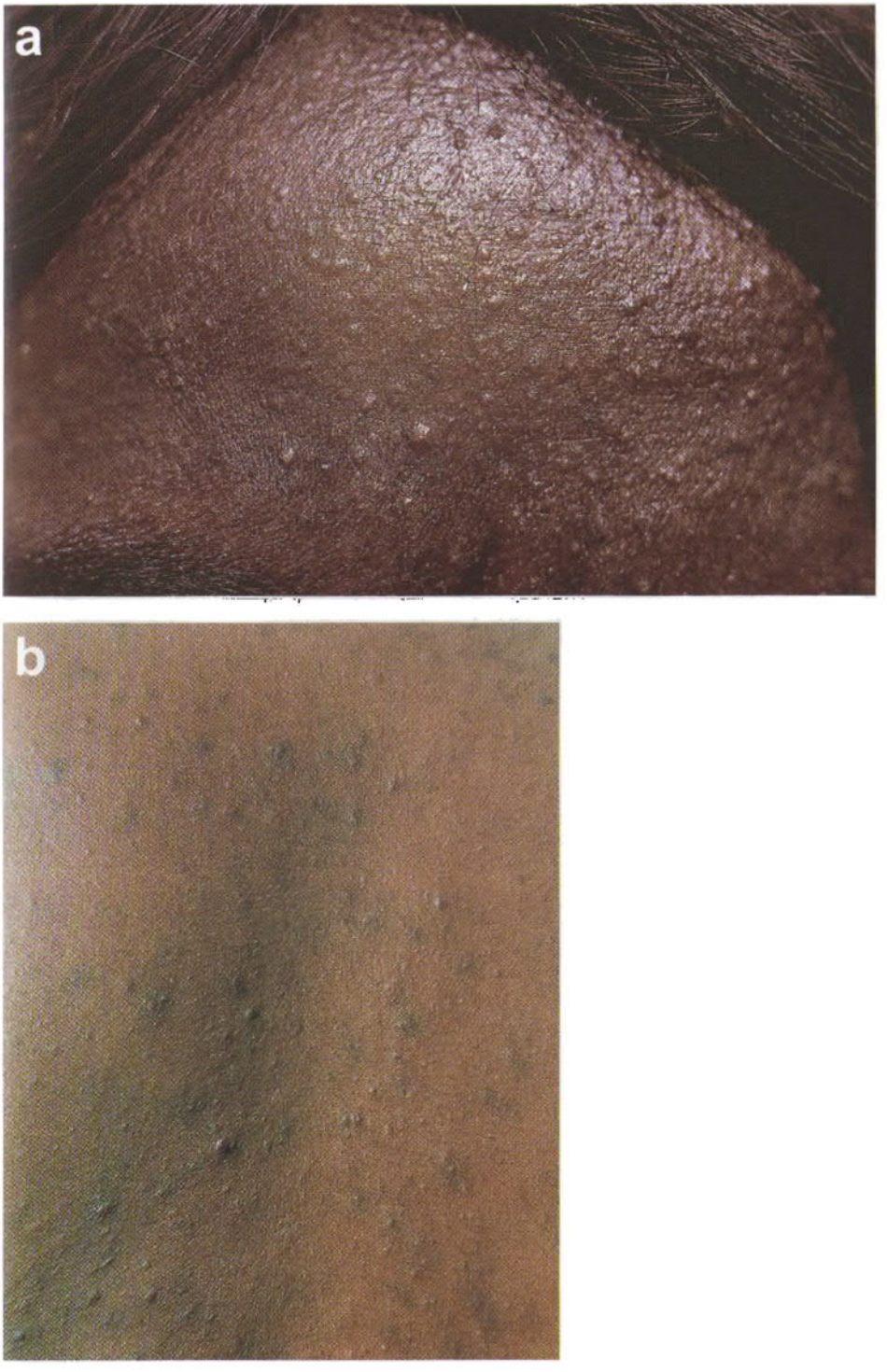
- Complex;
- Diet is unlikely to be a significant factor.

Making the diagnosis

- Clinical.

Significance

- Acne and post-acne scarring can affect self-esteem and is regarded as a risk factor for unemployment and suicide.



a *Comedonal acne on the forehead.*

b *Predominantly comedonal acne with a few papules and early pustules on the back.*

Lip Biting, Chewing, Licking (perleche), Picking and Sucking

Summary: Traumatic changes on the lips and inside the mouth, which may need to be distinguished from inflammatory skin diseases.

CLINICAL DESCRIPTION

- May involve lips and biting the inside of the cheeks;
- Redness;
- Lightening of colour (hypopigmentation);
- Scarring may result;
- Inside the cheek (buccal mucosa) produces a white line where the teeth bite (keratinisation).

Epidemiology

- Common;
- Most common in young males.

Causes

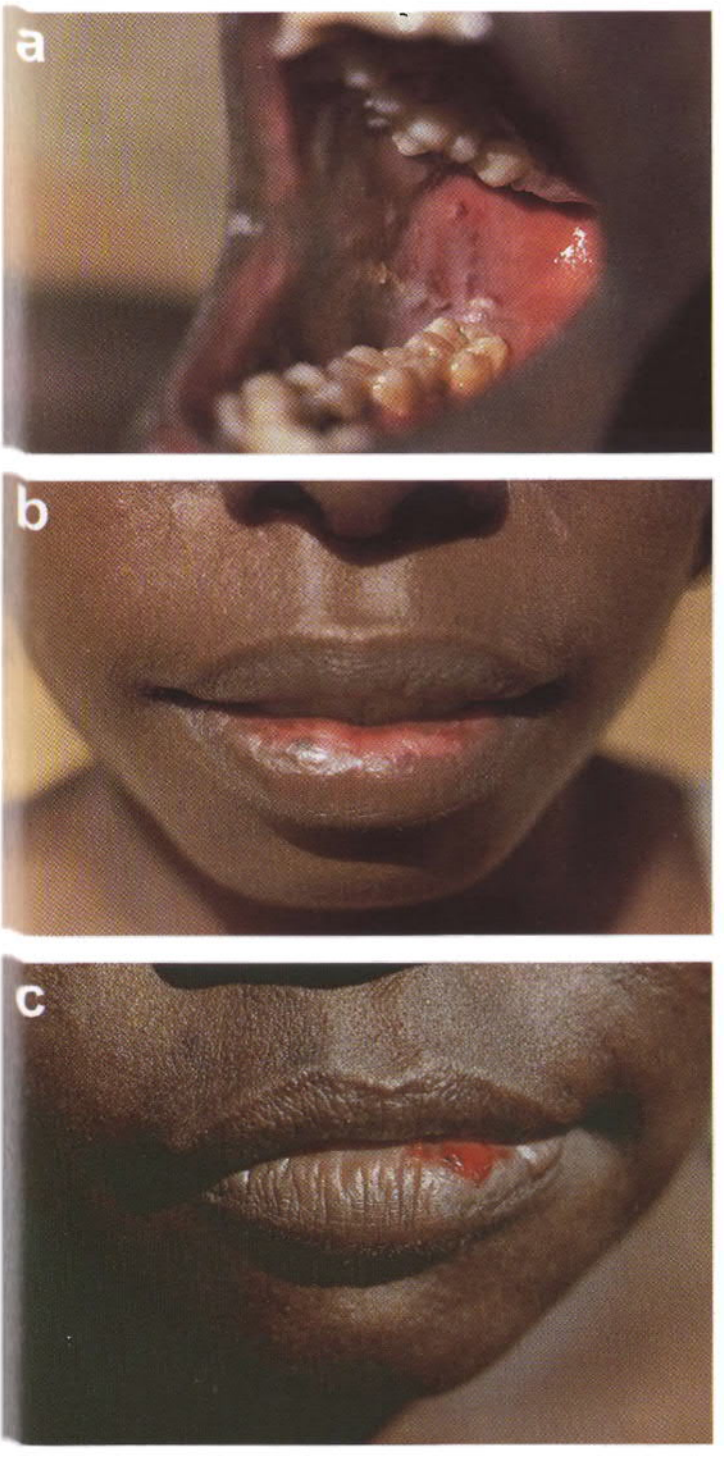
- Habit;
- Reason given may include the lips feel too dry or too much sun;
- Similar changes may be seen following injuries from fighting.

Making the diagnosis

- The habit may be observed;
- May need to distinguish from discoid lupus erythematosus (DLE) of the lip, especially in females (discussed further on page 48);
- Cheek biting should be distinguished from oral lichen planus (rare in Aboriginal populations) – linear rather than lace-like network, only along the line where the teeth bite. Check for signs of lichen planus elsewhere in mouth, skin, scalp and nails.

Significance

- Is of no significance; only for differential diagnosis.



a *Keratinisation along the bite line of buccal mucosa.*
b *Lip sucker.*
c *Lip biter with recent trauma.*

Chewing Tobacco Mucositis

Synonym: Pitcheri (pitjuri) mucositis and other local language names.

Summary: Inflammation of the inside of the mouth due to the irritant effects of holding a quid of chewing tobacco for long periods.

CLINICAL DESCRIPTION

- Affects inside cheek (buccal mucosa) or inner aspect of lower lip;
- Location depends on where the individual holds the quid of chewing tobacco between the teeth and gums and either inside of cheek or lower lip;
- Tobacco quid may also be held on the lower lip;
- Usually oval, 2–3 cm long and generally to one side, but can be bilateral;
- Inflammatory reaction – red (erythema);
- With or without epithelial necrosis – white, erosions;
- Does not apparently undergo malignant change.

Epidemiology

- Chewing tobacco is used by both sexes – female predominance 38%, males 11% in Northern Territory;
- Now mainly seen in the elderly.

Cause

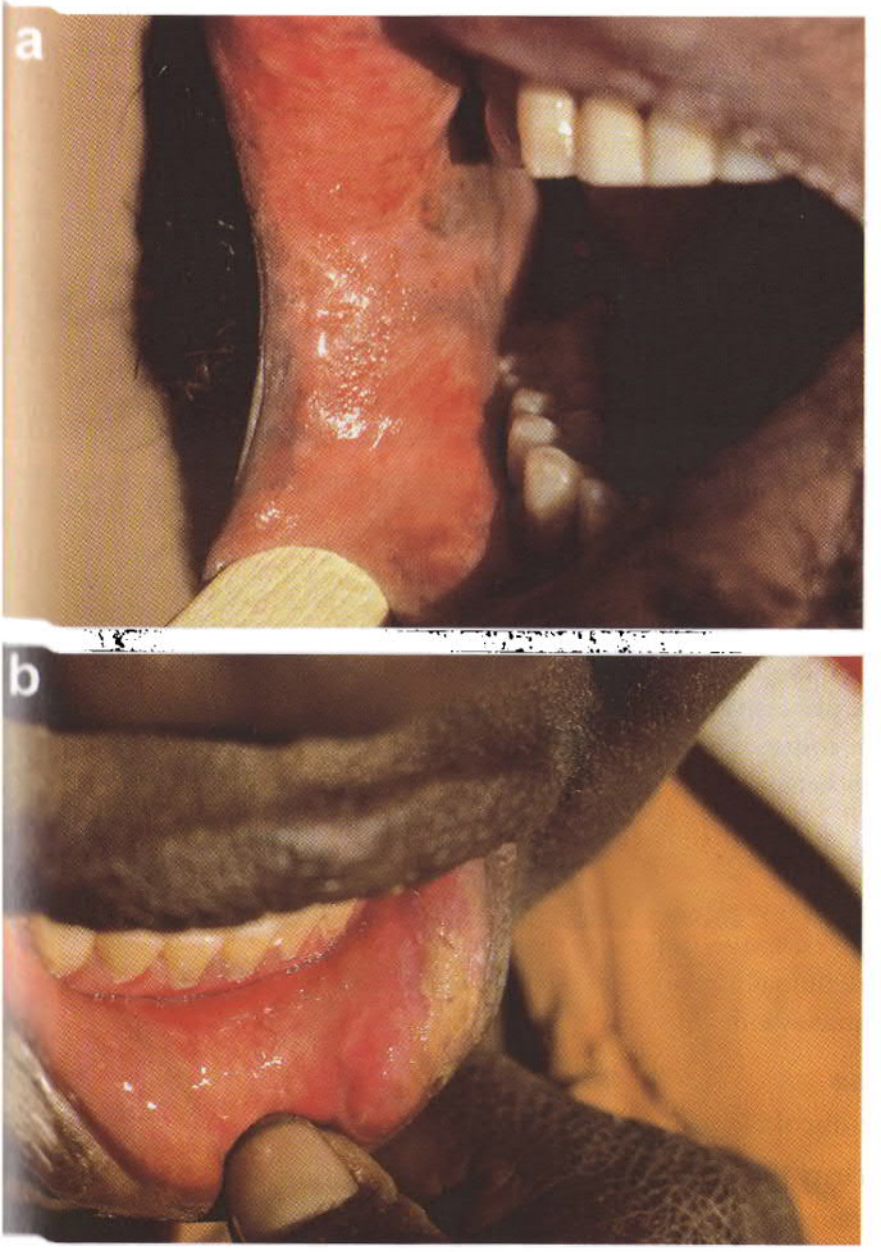
- Sucking or chewing a quid (ball or lozenge-shaped mass) of commercial tobacco or native tobacco prepared from *Duboisia hopwoodii* or several species of *Nicotiana*.

Making the diagnosis

- Habit is usually observed;
- Tobacco quid may be observed held behind the ear;
- May need to exclude other inflammatory diseases of the oral mucosa such as discoid lupus erythematosus (DLE) or lichen planus – history; clinical examination including scalp, face, general skin examination; biopsy.

Significance

- If due to commercial tobacco, there may be dental caries and nicotine-related adverse effects.



a *Chewing tobacco mucositis of buccal mucosa.*
b *Chewing tobacco mucositis of the inner aspect of the lower lip.*

Discoid Lupus Erythematosus (DLE) of the Lip

Summary: Inflammatory condition of the lips, most commonly the lower, which may be associated with systemic disease, and in which squamous cell carcinoma (SCC) may develop at a late stage.

CLINICAL DESCRIPTION

- Most commonly affects the lower lip, sometimes the upper, occasionally both;
- Can be restricted to the lip alone, but other sites can be affected (see sections on face (page 35), scalp);
- Acute stage – red, friable, granular, bleeds easily, may be covered with clot or crust;
- Subacute – superficial erosion or ulceration, crusting or maceration. Shallow ulcers often bordered by sodden white epithelium with secondary infection by *Candida albicans*;
- Chronic – loss of pigment with or without scarring;
- Five per cent undergo malignant change to SCC;
- The course is long and punctuated by periods of improvement with healing and deterioration or recurrence.

Epidemiology

- Common in the Aboriginal population;
- More common in females than males;
- Onset usually between puberty and late forties;
- Australia-wide.

Cause

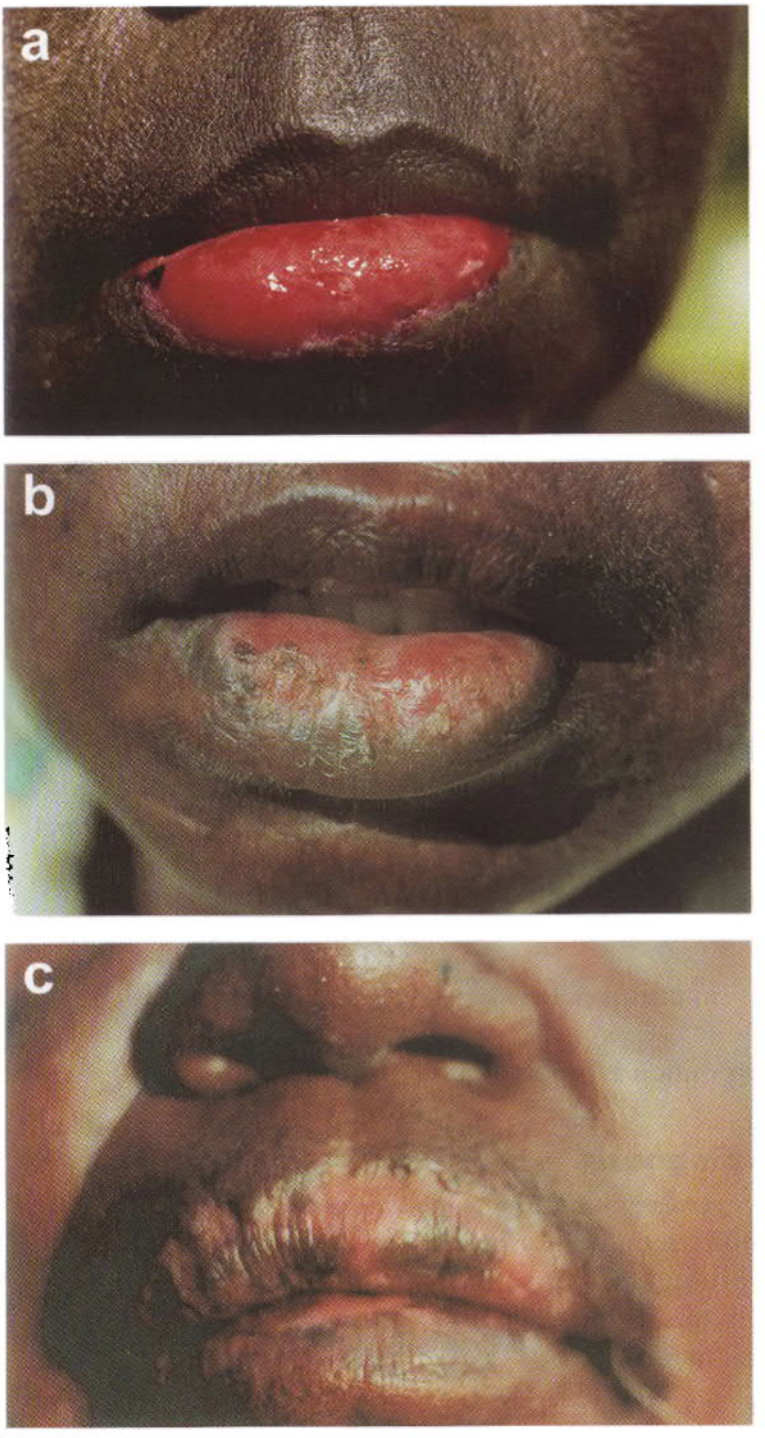
- See discoid lupus erythematosus (DLE) of the face (see page 35).

Making the diagnosis:

- Examine other possible sites of involvement (e.g. scalp, face);
- Requires biopsy for histology and immunofluorescence;
- May be misdiagnosed as lip biting, chewing tobacco mucositis, syphilis, candidosis, herpes simplex or squamous cell carcinoma.

Significance

- May be associated with systemic lupus erythematosus;
- Important to monitor for the development of SCC of the lip.



a *Acute discoid lupus erythematosus (DLE) of the lower lip.*
b *Acute to subacute DLE of the lower lip.*
c *Chronic DLE involving both the upper and lower lips.*

Focal Epithelial Hyperplasia

Synonym: Heck's disease.

Summary: Benign oral papules and polyps.

CLINICAL DESCRIPTION

- Single or more usually multiple painless polyps;
- Mainly on inside cheek (buccal) and inside lip (labial) mucosa
  – Can involve the tongue and gums;
- Ovoid, sessile, soft, raised (papules);
- Slightly paler than the surrounding mucosa;
- Usually discrete;
- Can be clustered or become confluent;
- Most are approximately 2.0 mm in diameter, ranging up to 8.0 mm;
- Large papules show a granular surface stippled with red dots;
- Redness (erythema) or firmness (induration) of the base is lacking.

Epidemiology

- Considered rare;
- Reported in 5.2% of Aborigines aged 4–25 years examined in central Australia. Lesions found in those aged 5–17 years (Williamson, pers. comm., 1970).

Causes

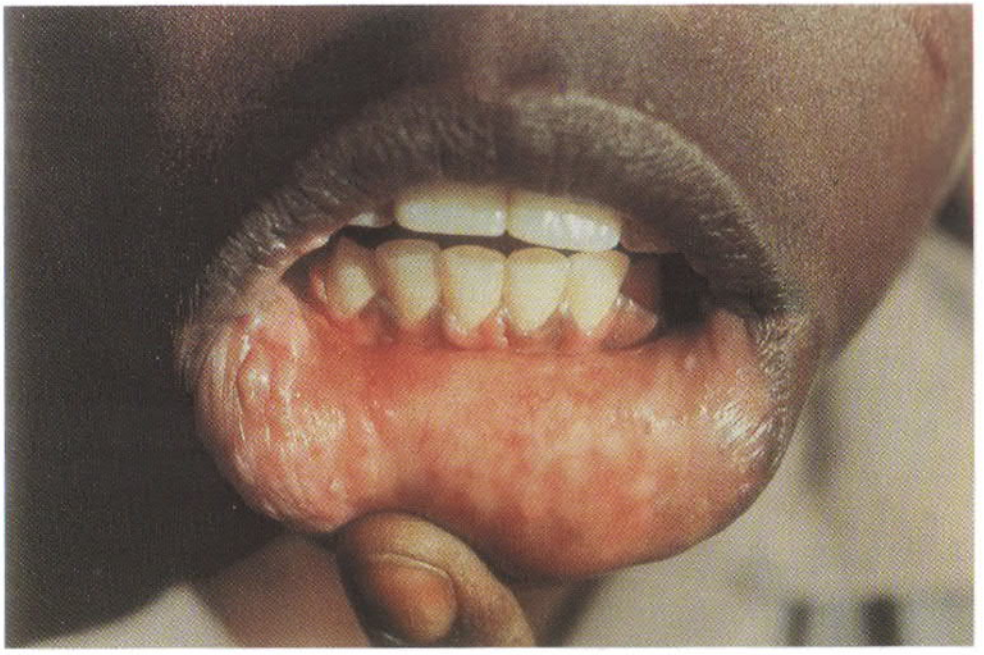
- Possibly viral – human papillomavirus types 13 and 32 have been implicated;
- Genetic predisposition as reported most commonly in indigenous populations.

Making the diagnosis

- Histology – hyperplasia with parakeratosis, acanthosis, dyskeratosis, numerous mitoses, focal cellular necrosis, small clear vacuoles in many nuclei of the prickle cells but no inclusion bodies. Loosely textured fibrous tissue in the core of the polyp; melanin more conspicuous in adjacent oral mucosa.

Significance

- Benign. Is of no clinical significance;
- May need to distinguish from mucosal papules of lepromatous leprosy.



Focal epithelial hyperplasia of the lower lip and gum.

Residual Ochre

Summary: Ochre staining of the scalp is externally applied dyestuff for decorative purposes and has no clinical significance, but may be confused by health professionals as a form of pathology.

CLINICAL DESCRIPTION

- Scaly scalp;
- Remains of decorative ochres.

Epidemiology

- Restricted to regions of Australia where ochres are used.

Cause

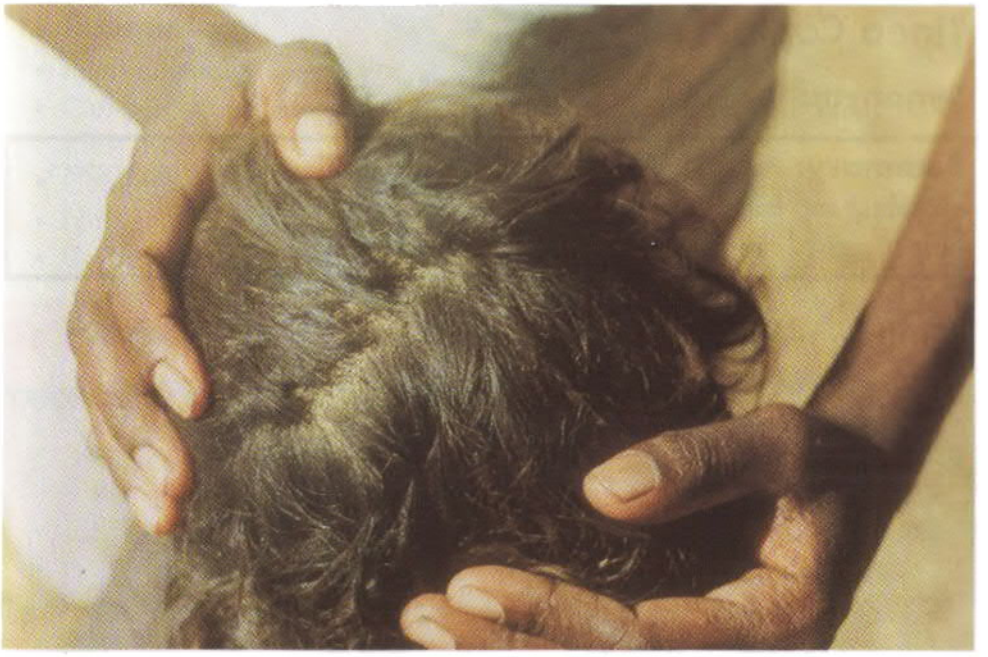
- Use of earth consisting of a mixture of clay and hydrated iron oxides for decorative cultural practice for ceremonies.

Making the diagnosis

- History;
- Not psoriasis, seborrhoeic dermatitis or dandruff, which are rarely seen in Aboriginal Australians.

Significance

- None. Important not to mislabel as psoriasis or ringworm of the scalp.



Remains of decorative ochres in the scalp.

Tinea Capitis

Synonym: Ringworm of the scalp.

Summary: Fungal infection of the scalp presenting as varying degrees of scaling and hair loss, which may become secondarily infected (impetiginised) and cause scarring hair loss.

CLINICAL DESCRIPTION

- It is not possible to determine the causative organism on clinical features alone;
- Usually poorly defined, scattered areas of white scale with varying degrees of hair loss;
- Scale may be diffuse or defined, mild (dandruff-like) or thick and adherent;
- Hair loss may be apparently absent, mild, severe or due to broken hairs (black dots).
- Kerion (inflammatory boggy folliculitis) rare in Aboriginal populations.

TRICHOPHYTON VIOLACEUM

CLINICAL DESCRIPTION

- Initial lesion – rarely seen – inconspicuous and fleeting, solitary, irregularly circinate lesion, faintly erythematous, slightly raised and scaly border;
- Usually non-inflammatory – finely scaly in scalp (and rarely beard);
- Diffuse, ill-defined – may be misdiagnosed as dandruff;
- Usually multiple areas involved;
- Hair loss may be absent, minimal (occasional broken hairs seen on close examination only), more apparent scattered broken hairs or obvious partial alopecia;
- Can be inflammatory, but rarely severe;
- Black dot tinea capitis – very short broken hairs, 'swollen black stump broken off level with or just below the mouth of the follicle' – of limited clinical value;
- Does not fluoresce under Wood's light;
- Varying degree of permanent scarring is a very common end-result.

Epidemiology

- Predominantly in children;
- Can persist into adult life – sometimes in adult females but rarely in adult males;
- Common cause in central and southern Australia;
- Not seen/rare in northern Australia;
- Common cause of tinea capitis in Aboriginal children in South Australia – mainly/only from areas close to the coastline;
- May coexist with *T. tonsurans*.

TRICHOPHYTON TONSURANS

CLINICAL DESCRIPTION

- Characteristically poorly defined and diffuse, mild scale, non-inflammatory;
- Patchy hair loss;
- Scale may be adherent;
- Black dot tinea – short broken hairs – of limited clinical value;
- Does not fluoresce under Wood's light.

Epidemiology

- Predominantly in children, sometimes in adult females but not adult males;
- Common cause of tinea capitis in Aboriginal children in central and South Australia;
- Not seen/rare in northern Australia;
- Commonest cause of tinea capitis in Aboriginal children in South Australia – distributed throughout the state.

MICROSPORUM CANIS

CLINICAL DESCRIPTION

- Similar to *Trichophyton* endothrix tinea capitis;
- Poorly defined, fine white scale or diffuse white finely scaly patches scattered over a wide area of the scalp;
- Minimal inflammation except in cases with secondary bacterial infection;
- Minimal hair loss;
- Scattered broken hair stubs in these patches show characteristic blue-green-yellow fluorescence under Wood's light, which may be more obvious on pulled hairs;
- May be very mild, resembling dandruff – non-inflammatory, fine white scaling, poorly defined with minimal inflammation or hair loss;
- Infected hairs may appear grey and lustreless.

Epidemiology

- Most common cause of tinea capitis in Australian Caucasian children, but generally not in Aboriginal children;
- However, in some Aboriginal communities *M. canis* variants may be very common in cats, dogs and children,
- Seen predominantly in children.

Causes

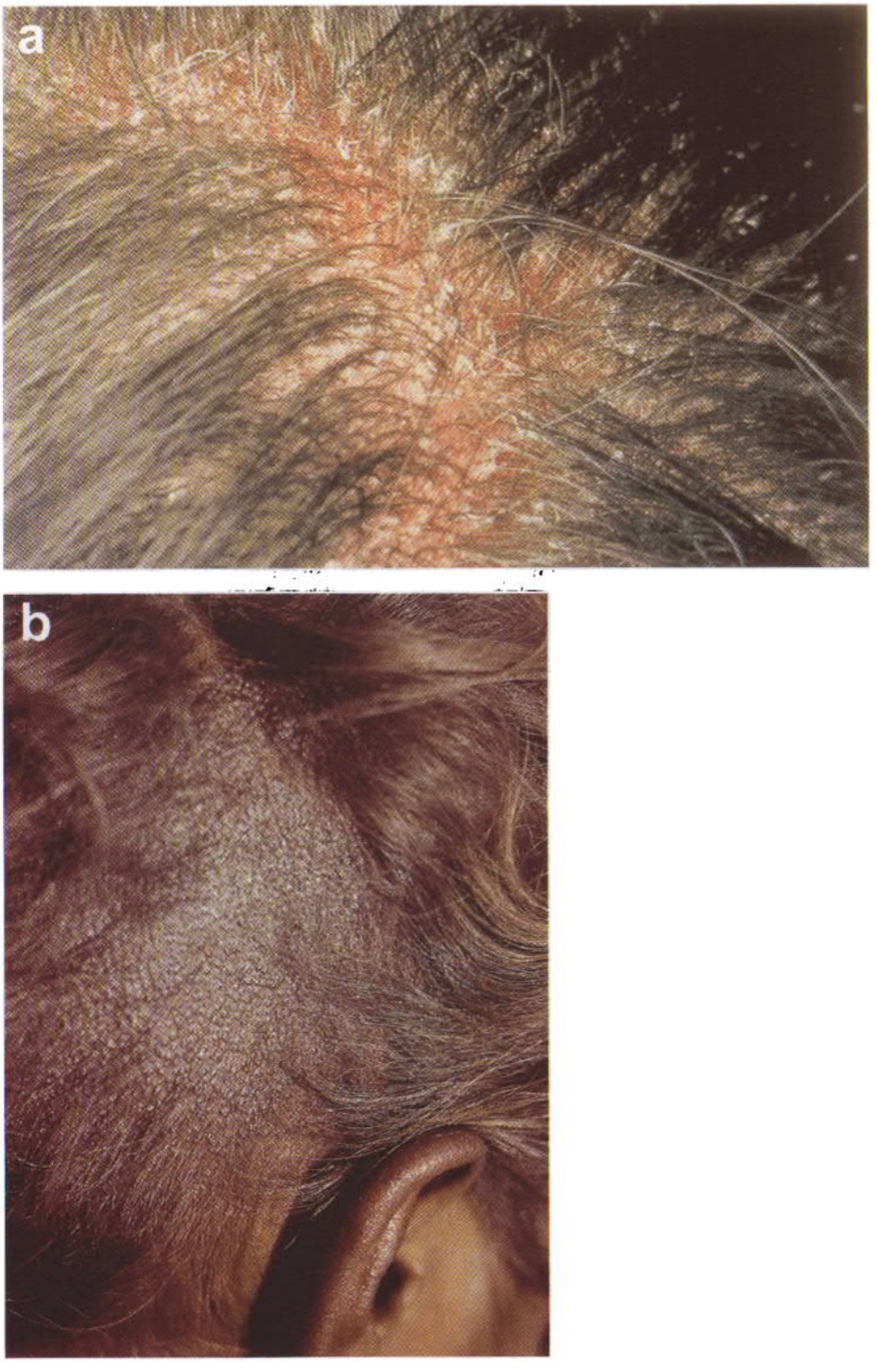
- The *Trichophyton* species listed are anthropophilic (human source) fungi – endemicity reflects overcrowding;
- *Microsporum canis* is zoophilic from cats and dogs – close contact with dogs and cats;
- Other dermatophytes that may cause tinea capitis less commonly include *Trichophyton verrucosum* (cattle), *T. mentagrophytes* (kangaroos) and *M. gypseum* (soil);
- *Trichophyton rubrum* (granular variant) – although the granular variant is endemic among Aboriginal populations in high rainfall, humid, tropical northern Australia, it is an unusual/rare cause of tinea capitis.

Making the diagnosis

- Important to re-examine the scalp after treatment for secondary impetiginisation as there may be underlying tinea;
- May be suspected when lesions on other sites (glabrous skin) are noted;
- Wood's light examination – positive with *M. canis* infection;
- Adequate skin scraping from the scalp (brush technique) and pulled hairs required for fungal microscopy/culture and diagnosis (discussed further on page 23);
  - Hair microscopy:
    M. canis – small spores, ectothrix (spores form a sheath around the surface of the affected hair shaft);
    T. tonsurans and *T. violaceum* – large spores, endothrix (hyphae penetrate the hair shaft and break up into parallel chains of arthrospores);
    T. rubrum – ecto-endothrix (spores both within the hair shaft and outside of it).
  - Specimens will remain viable for up to 30 days and so can be suitably packed and sent to a distant laboratory for culture;
  - Specimens can be spread on culture media (slopes or small plates) directly.

Significance

- Secondary bacterial infection (impetiginisation).



a Trichophyton violaceum *of the scalp.*
b Trichophyton tonsurans *was isolated.*

Head Lice

Synonyms: Nits, pediculosis capitis.

Summary: Often endemic infestation of the hair by the human head louse, which commonly becomes secondarily infected (impetiginised).

CLINICAL DESCRIPTION

- Common sites to find eggs ('nits') in scalp are above the ears and the nape/occipital hair line;
- Any hairy site may be infested;
- Enlarged cervical lymph nodes should prompt examination of the scalp hairs for eggs ('nits');
- Adult head lice can be difficult to see on the scalp rather than on hairs;
- Eggs ('nits') are firmly attached to the hair and cannot be easily pulled along the hair;
- Infected excoriations of the scalp;
- Secondary bacterial infection is very common.

Epidemiology

- Endemic in some Aboriginal communities with up to 90% of children infested.

Cause

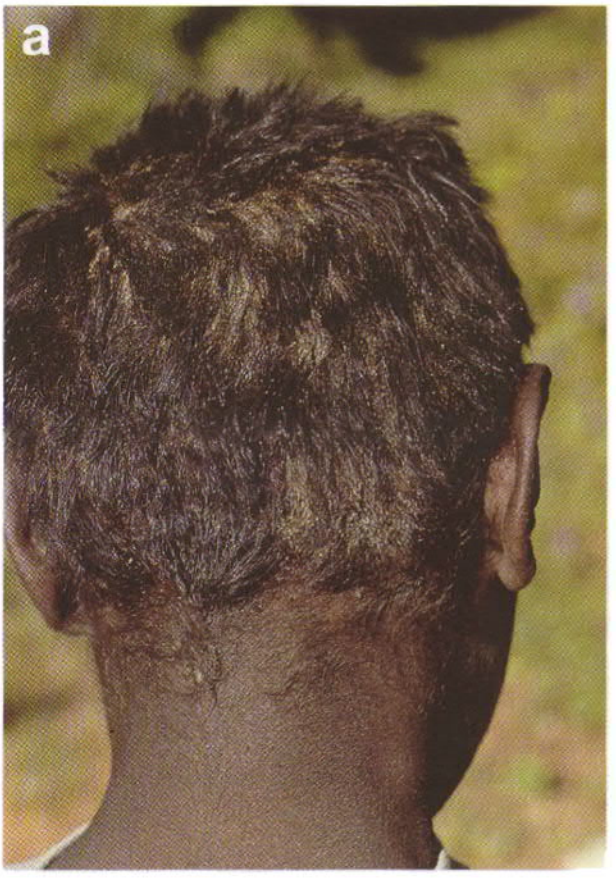
- *Pediculus humanus capitis* (human head louse).

Making the diagnosis

- Usually clinical
  - Adult lice can be difficult to see as they are mobile and move quickly out of the light when exposed by separating the hairs;
- Plucked hairs – microscopy for eggs;
- 'Pseudo-nits' – can easily be pulled along a hair and are usually skin scales;
- Important to re-examine the scalp after treatment of impetigo as this may have been secondary to underlying head lice.

Significance

- The endemicity of infestation is a marker of poor socio-economic circumstances and overcrowded living conditions of many Aboriginal people. Infestation may result in secondary bacterial infection.



a *Excoriated, infected pediculosis capitis with enlarged cervical lymph node on the right side.*

b *Eggs ('nits') on scalp hairs.*

Trichomycosis Axillaris

Summary: Benign infection of axillary hairs, which may be of concern because of effects on personal odour or appearance.

CLINICAL DESCRIPTION

- Concretions/encrustations/nodules/sheaths on hairs;
- Most commonly yellow, occasionally red, rarely black;
- Usually armpit (axillary) hair, rarely pubic;
- Can be unilateral or bilateral, localised to the dome of armpit or diffuse;
- Proximal hair (closest to the skin surface) most frequently and heavily affected;
- Affected hairs may be brittle;
- Usually asymptomatic;
- Sweat may also be discoloured, staining clothing – may be intermittent;
- May be associated with odour;
- No changes in axillary skin;
- Wood's lamp examination – fluoresce yellow or blue-white.

Epidemiology

- Very common;
- Hot humid conditions;
- Affects both sexes.

Causes

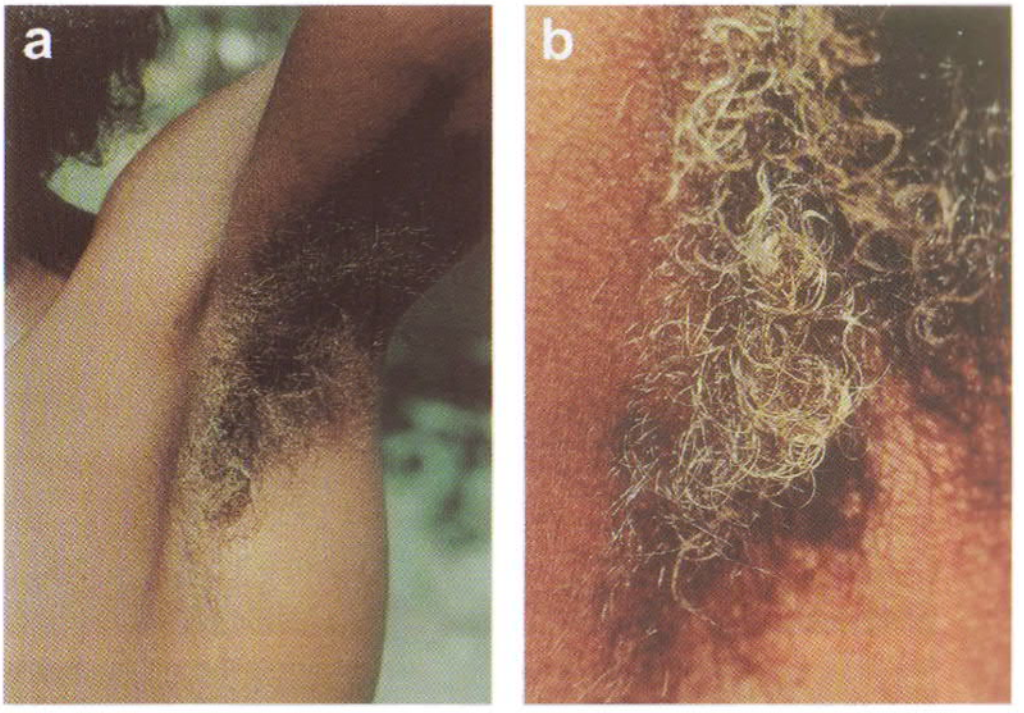
- Variety of aerobic Corynebacteria – normal axillary bacterial flora;
- Does not relate to group, age, hygiene or degree of sweating or hairiness.

Making the diagnosis

- Usually clinical;
- May need to be distinguished from lice or powder;
- Microscopy of concretion – tightly packed, narrow Gram-positive bacilli;
- Culture: Pluck hair, immerse in 70% alcohol, then incubate on blood agar at 37°C.

Significance

- Cosmetic only; not to be confused with lice.



a *Trichomycosis axillaris of the armpit (axilla).*

b *Close examination of the axillary hairs will reveal the concretions.*

Madarosis

Summary: Loss of eyebrows, which may be benign or an important sign of lepromatous leprosy or other systemic diseases.

CLINICAL DESCRIPTION

- Partial or complete loss of eyebrows;
- Most commonly affects the outer two-thirds of the eyebrow.

Epidemiology

- Seen in both sexes but predominantly in females;
- Familial form seen;
- Onset occurs in late 30s.

Causes

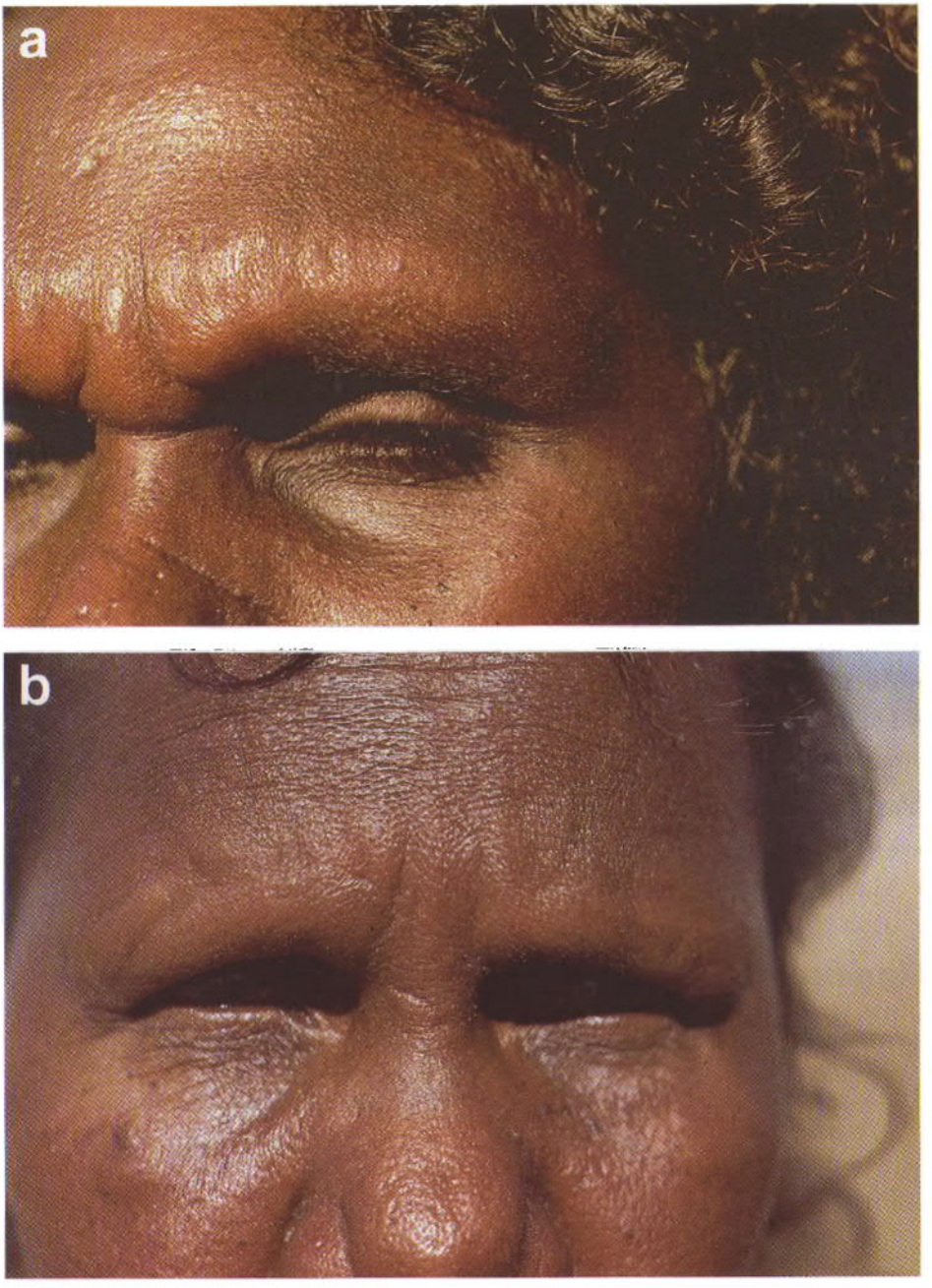
- Genetic;
- Trauma – plucking, rubbing, burns;
- Infections – lepromatous leprosy, secondary syphilis;
- Thyroid disease.

Making the diagnosis

- Familial form needs to be distinguished from other causes of loss of eyebrows, usually on history and general clinical examination.

Significance

- A benign entity unless due to pathology or of cosmetic concern.



a *Familial loss of eyebrows.*
b *Loss of eyebrows in lepromatous leprosy.*

Traumatic Scarring Alopecia

Summary: Permanent hair loss following accidental or deliberate injury.

CLINICAL DESCRIPTION

- Permanent hair loss;
- Loss of hair follicles – no hair openings visible, smooth shiny skin;
- Usually depigmented (loss of pigment);
- May show a linear pattern, reflecting the inflicted injury;
- Sorry cuts are on the vertex and crown of scalp.

Epidemiology

- Sorry cuts are self-inflicted, mainly by women, at the death or burial of a relative or friend.

Causes

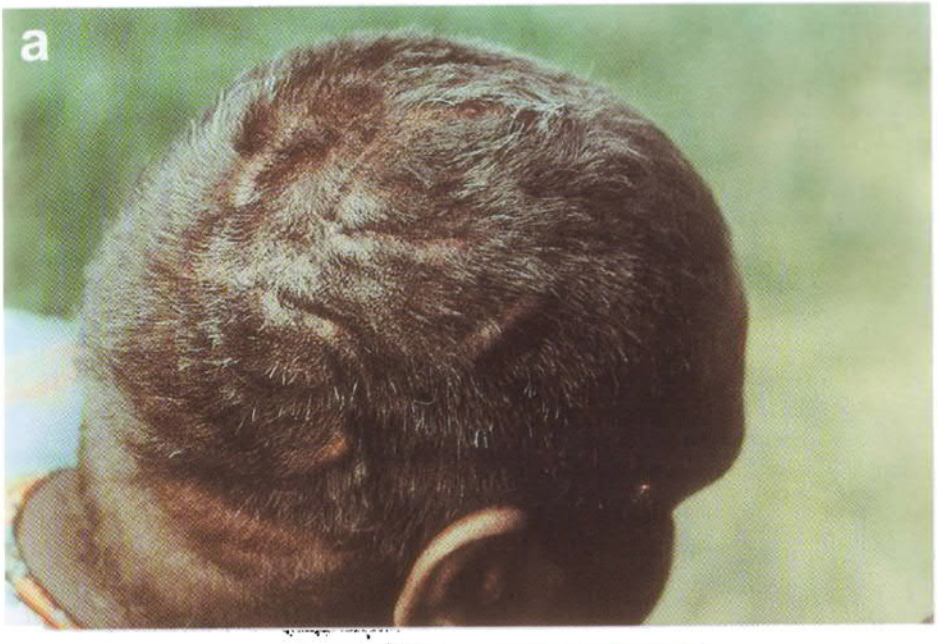
- Accidental – burns, lacerations;
- Deliberate – sorry cuts – using a stone, club, bottle or other blunt instrument.

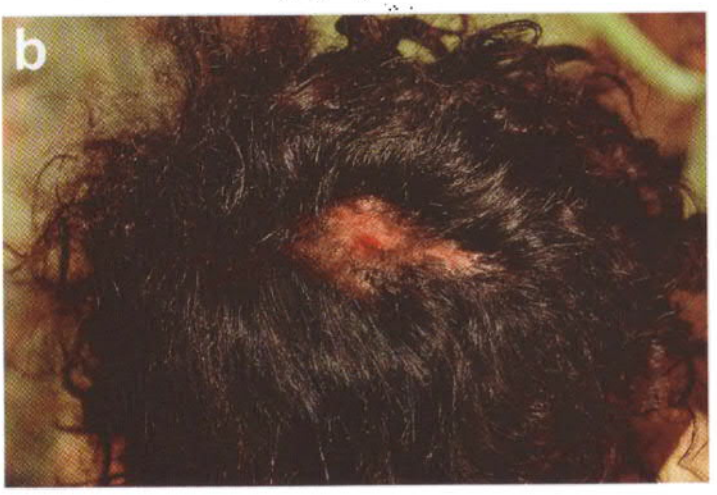
Making the diagnosis

- Usually on history;
- May need to distinguish from discoid lupus erythematosus (DLE) of the scalp.

Significance

- None, but important to exclude other causes of scarring hair loss.





a *Healed lacerations of the scalp.*

b *Scarring alopecia after sorry cuts on the vertex of the scalp.*

Discoid Lupus Erythematosus (DLE): Scarring Alopecia

Summary: Early inflammatory lesions result in permanent hair loss, which may be associated with systemic disease.

CLINICAL DESCRIPTION

- Permanent localised hair loss;
- Usually of the scalp;
- Can occur anywhere on the scalp;
- May be multiple and scattered;
- Associated loss of pigment;
- May see early/active inflammatory lesions with redness (erythema) and scale, and able to easily pull hairs out at the edges (positive hair tug test).

Epidemiology

- See DLE of the face (see page 35).

Cause

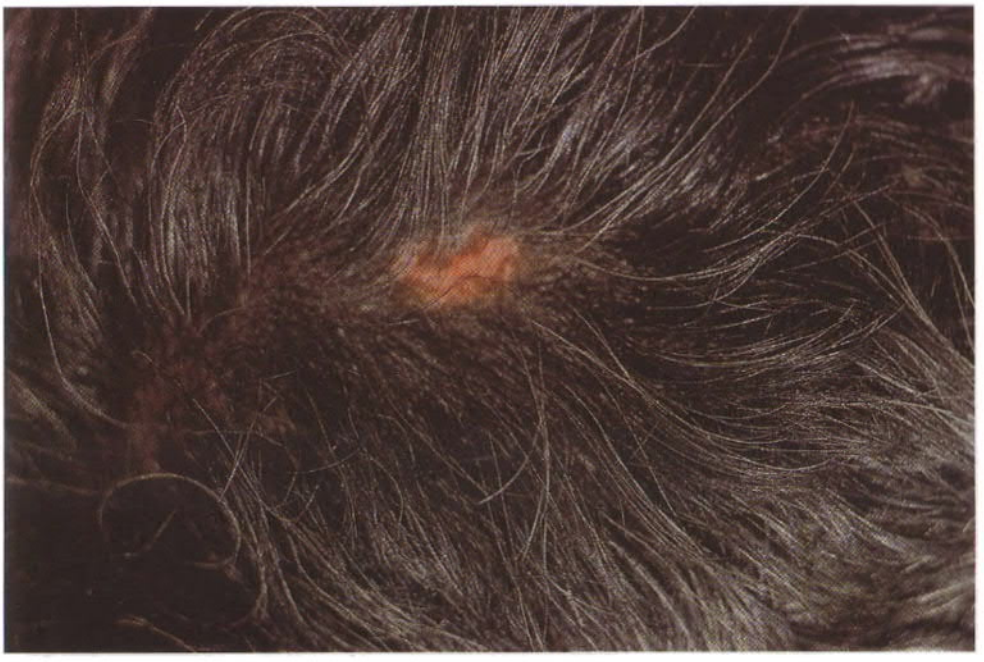
- See DLE of the face (see page 35).

Making the diagnosis

- Clinical examination – for signs of DLE elsewhere;
- May need to distinguish from traumatic scarring alopecia;
- Scalp biopsy for histology: Two 4 mm punch biopsies from inflammatory edges, in 10% buffered formalin, allows horizontal and vertical sectioning by the pathologist.

Significance

- Important to investigate for systemic lupus erythematosus.



Chronic discoid lupus erythematosus (DLE) of the scalp resulting in localised scarring alopecia.

Primary Syphilis

Synonym: Great pox.

Summary: A predominantly sexually transmitted disease presenting in the ano-genital area as a painless self-healing ulcer which, if untreated, may progress to secondary and tertiary stages.

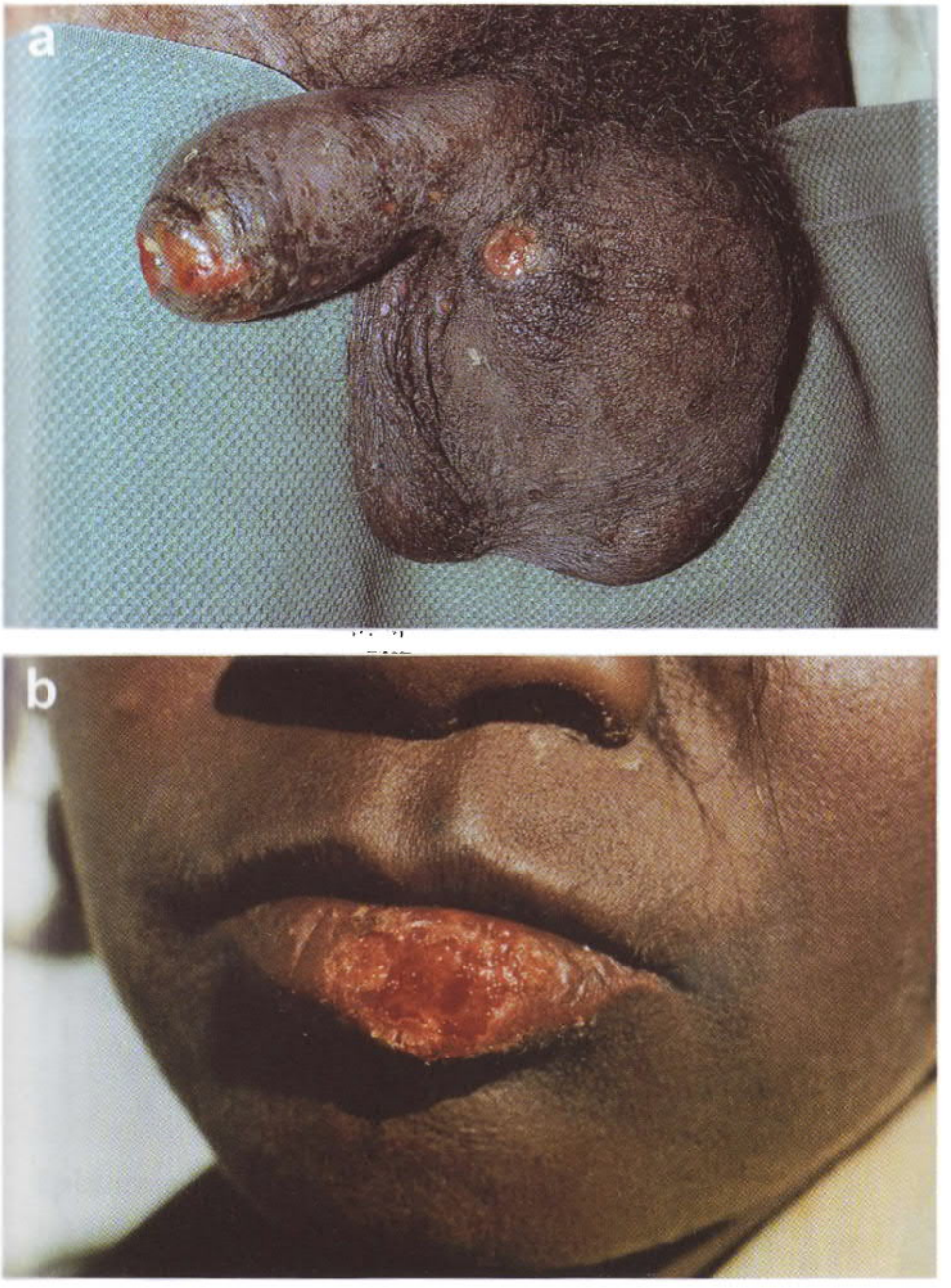
CLINICAL DESCRIPTION

Primary Chancre(s)

- Develops at site of inoculation;
- Usually single but can be multiple;
- Usually in genital area;
  Other sites:
  – Anus, rectum
  – Lip, tongue, tonsil, eyelid
  – Finger;
- Initially a small red spot that becomes raised then ulcerated in about 1 week;
- Round or oval in shape;
- Usually about 1 cm in diameter;
- Sharply defined edge, sometimes with a red halo;
- Feels firm like a button;
- Does not bleed easily;
- Asymptomatic unless secondarily infected;
- Swelling (oedema) may be considerable
  – of the urinary meatus may interfere with urination;
- Bilateral non-tender discrete inguinal lymph nodes palpable
  – Tender only with secondary infection;
- Anal chancre – painful indurated fissure;
- Heal spontaneously in 4–8 weeks, with or without scarring;
- May commonly pass unnoticed, especially in females;

Secondary stage

- May appear with the primary chancre still present.



a *Primary chancres of syphilis. (Reproduced with permission from the Royal Perth Hospital, Perth, WA, Australia.)*

b *Primary chancres of syphilis on the lower lip.*

Epidemiology

- Ninety per cent of reported cases of syphilis in Australia occur in Aboriginal communities and the incidence is decreasing;
- Transmission:
  - Predominantly sexually transmitted;
  - Less commonly transmitted by kissing;
  - Congenital infections (transplacental spread) can occur;
  - Blood contamination (e.g. needlestick injuries, sharing of needles);
  - Direct contact with open lesion.
- Incubation period average 3 weeks (range 9–90 days);
- Affects males and females.

Cause

- *Treponema pallidum* (*T. pallidum*), which is a corkscrew-shaped bacterium (spirochaete) with characteristic motility.

Making the diagnosis

- Demonstrate *T. pallidum* in fluid from the primary lesion or enlarged regional lymph node by dark ground microscopy or fluorescent antibody techniques if skilled staff and specialised equipment available;
- Saprophytic spirochaetes may be confused especially from mouth lesions;
- Serology: TPHA or EIA IgA and RPR with repeat testing 2–3 weeks later.

Significance

- Recognition permits early diagnosis and treatment to prevent spread to others and secondary and tertiary progression in the patient.

Donovanosis

Synonym: Granuloma inguinale.

Summary: A relatively uncommon genital ulcer disease with low infectivity but if untreated may result in extensive destructive chronic ulceration, which commonly becomes infected with anaerobic bacteria resulting in a characteristic and offensive odour.

CLINICAL DESCRIPTION

- Usually on genitalia (i.e. glans, prepuce, shaft) but sometimes on thigh, groin, perineum;
  – May occur on face (i.e. lips and nose);
- Early lesion – red, firm velvety smooth papule/nodule 1–3 mm or vesicle; painless, bleeds easily;
- May be solitary or several;
- Forms either a beefy-red granulomatous mass or a painless ulcer with a sharply defined overhanging edge;
- Regional lymph nodes are not enlarged;
- Spreads from edges of the lesions by continuity or auto-inoculation;
- Secondary infection leading to pain, offensive discharge and enlarged inguinal lymph nodes;
- Progression variable – may heal or extend rapidly or slowly, remissions and recurrences;
- Deep ulceration or epithelial hyperplasia, scarring, lymphoedema and deformity – all may occur;
- If extensive, cachexia may develop;
- Can spread to liver, spleen and bone;
- Typically present late with extensive and destructive lesions.

Epidemiology

- Mildly contagious;
- Endemic in remote northern and western regions
  – Thirty new cases per year reported Australia-wide;
- Probably sexually transmitted;
- Incubation period usually 1–4 weeks.

Cause

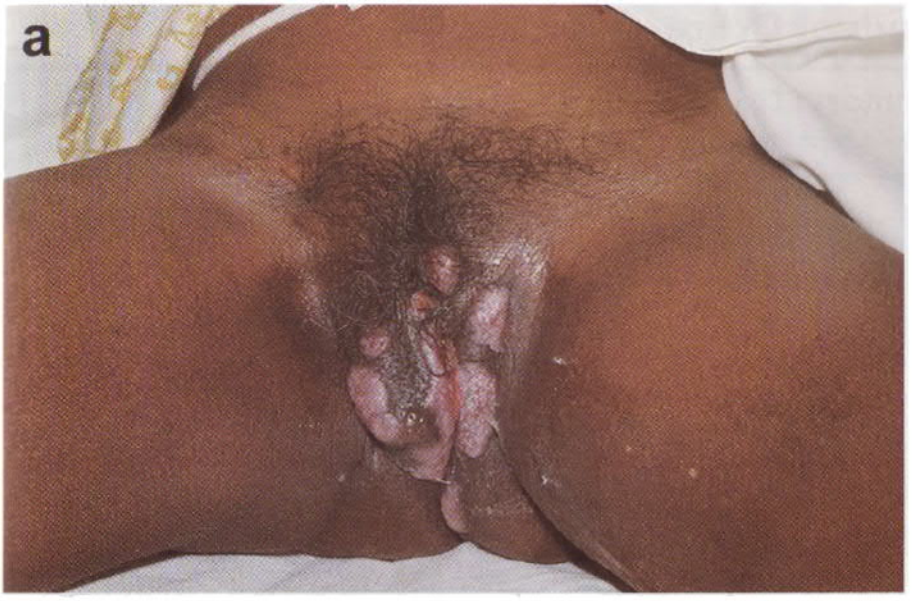
- *Calymmatobacterium granulomatis*, which is a Gram-negative oval bacillus.

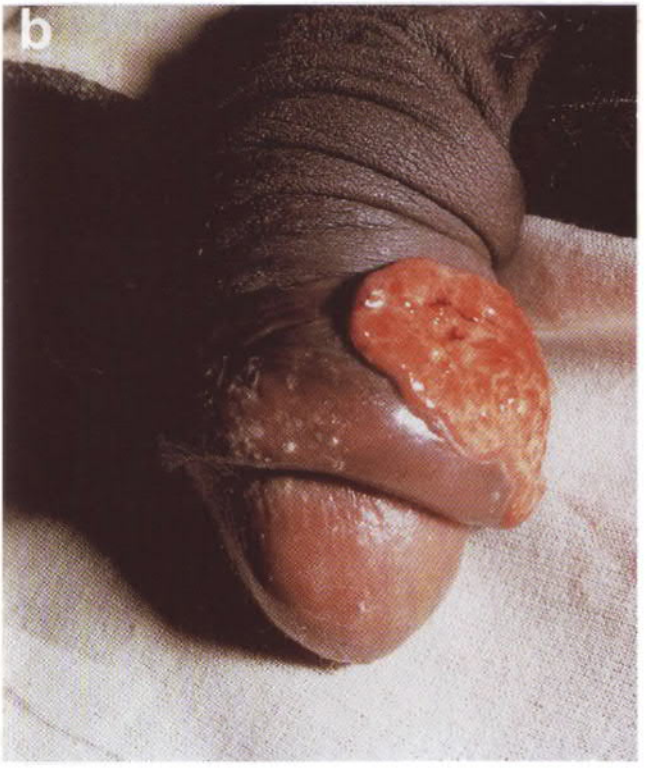
Making the diagnosis

- Need to exclude syphilis and other sexually transmitted diseases (STDs), which may be co-existing;
- Smear obtained from deep part of friable granulation tissue, pressed onto clean glass slide, air-dried or fixed in methyl alcohol, stained by Wright's or Giemsa stain. Look for Donovan bodies (i.e. bipolar bacillus in mononuclear cells);
- Tissue biopsy;
- Polymerase chain reaction (PCR) swab test;
- Organism difficult to culture.

Significance

- Chronic destructive infection that may be associated with systemic ill-health;
- Often coexists with other STDs.





a *Donovanosis in a female.*
b *Donovanosis in a male.*

Residual Ochre

Summary: The application of natural pigments to the skin for ceremonial cultural purposes.

CLINICAL DESCRIPTION

- Body painting using natural ochres;
- It washes off although is generally allowed to wear off.

Epidemiology

- Widespread throughout Australia where ochres are used;
- Styles of painting vary with tribal groups.

Cause

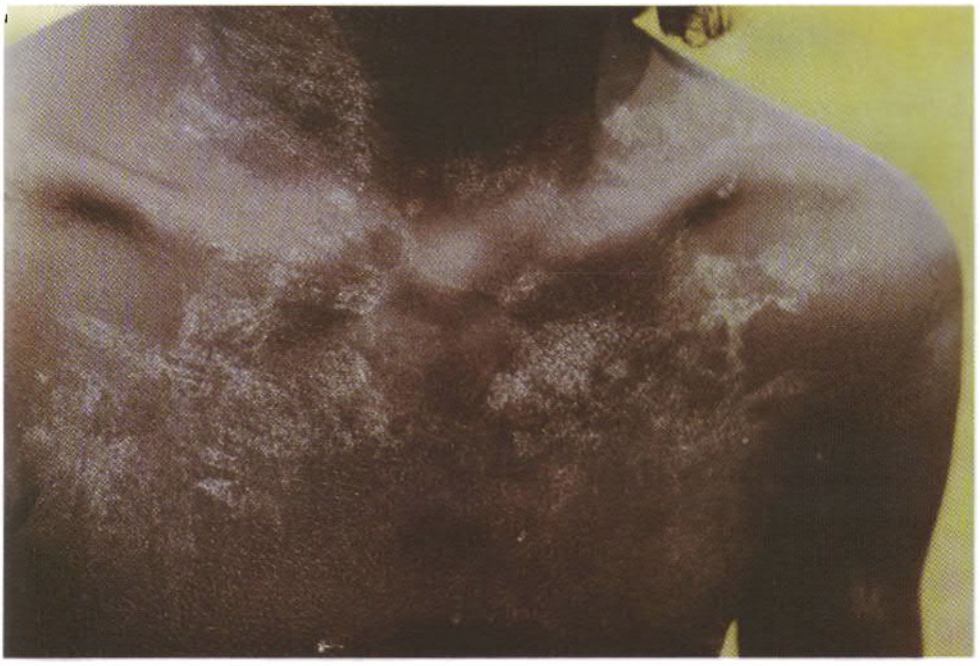
- Cultural – corroborees, initiations.

Making the diagnosis

- Diagnosis made easily on history;
- Not to be confused with pityriasis versicolor or tinea corporis (discussed further on pages 78 and 80).

Significance

- None, except may need to distinguish from infections.



Ochre remains on body.

Dry Skin

Synonym: Xerosis.

Summary: Scaly exposed skin due to the drying effects of the environment but which may need to be distinguished from systemic causes.

CLINICAL DESCRIPTION

- Superficially cracked epidermis in a pattern of dried mud pond or crazy paving but no associated thickening of skin;
- Mainly on exposed sites such as the face and legs.

Epidemiology

- Common in central Australia.

Cause

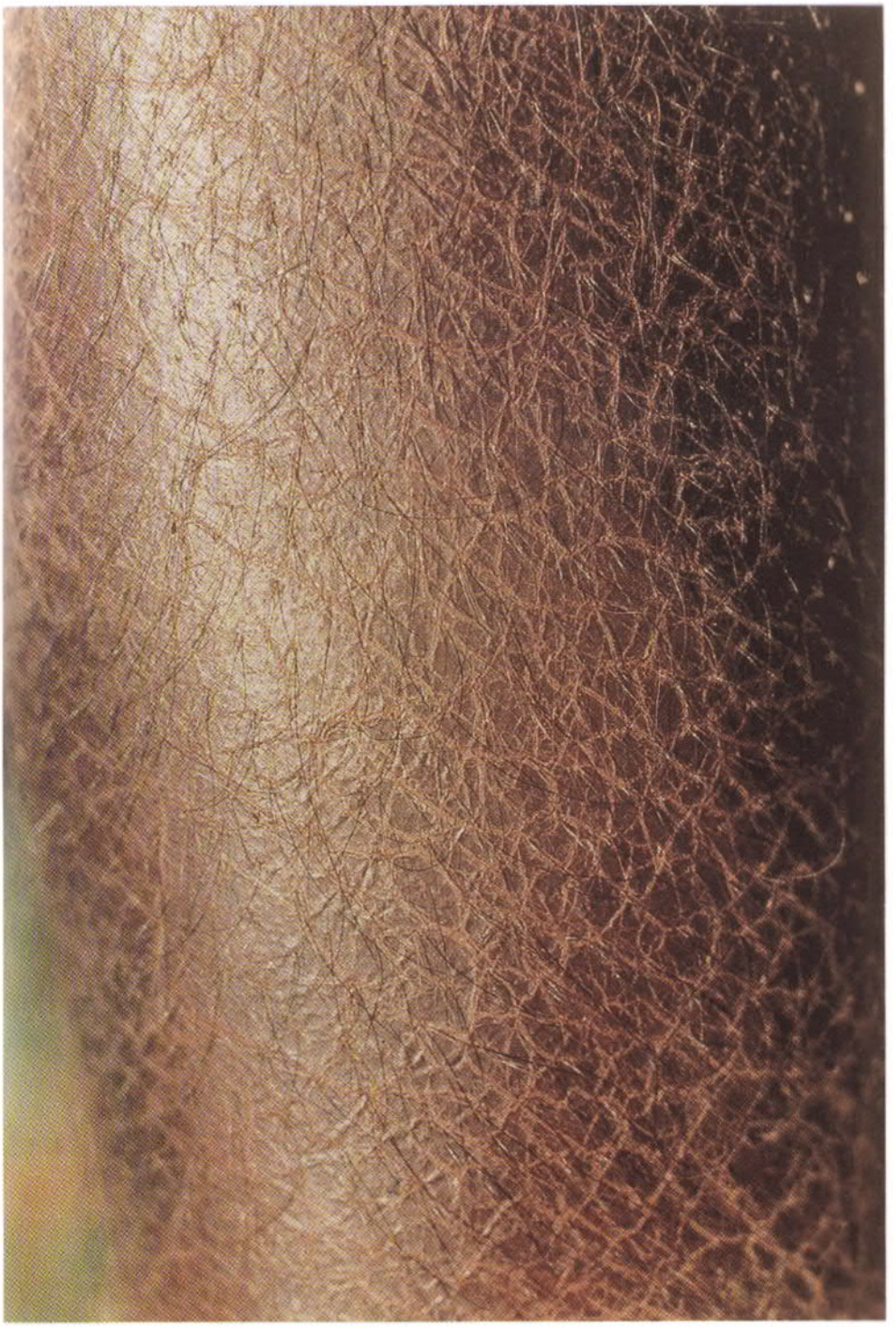
- Environmental – cold winds and weather, especially in winter months.

Making the diagnosis

- Not to be confused with ichthyosis (fish-scale), which may be inherited or acquired associated with systemic diseases (including leprosy) and medications;
- If very extensive may need to consider systemic causes of dry skin (e.g. thyroid disease).

Significance

- None but important to consider all forms of leprosy as a cause of dry or ichthyotic skin.



Dry scaly skin of the leg.

Pityriasis (Tinea) Versicolor

Synonyms: Darwin sunburn, white handkerchief.

Summary: Scaly hypopigmentation resulting from a yeast infection of cosmetic significance.

CLINICAL DESCRIPTION

- Light/pale-coloured (hypopigmented) scaly patches;
- Bilateral, reasonably symmetrical;
- Most common on upper trunk, upper arms, neck, occasionally on the face;
- Small and circular pattern – 'Darwin sunburn';
- Extensive sheets – 'white handkerchief';
- Scale is easily scratched off (grattinage; also see page 17);
- Usually asymptomatic;
- Wood's light examination – greenish-yellow fluorescence and the areas of reduced skin pigmentation are more evident;
- Commonly recurs after treatment.

Epidemiology

- Widely prevalent in hot humid regions;
- Usually between puberty and middle life, but has been seen even in infancy;
- Both sexes affected.

Causes

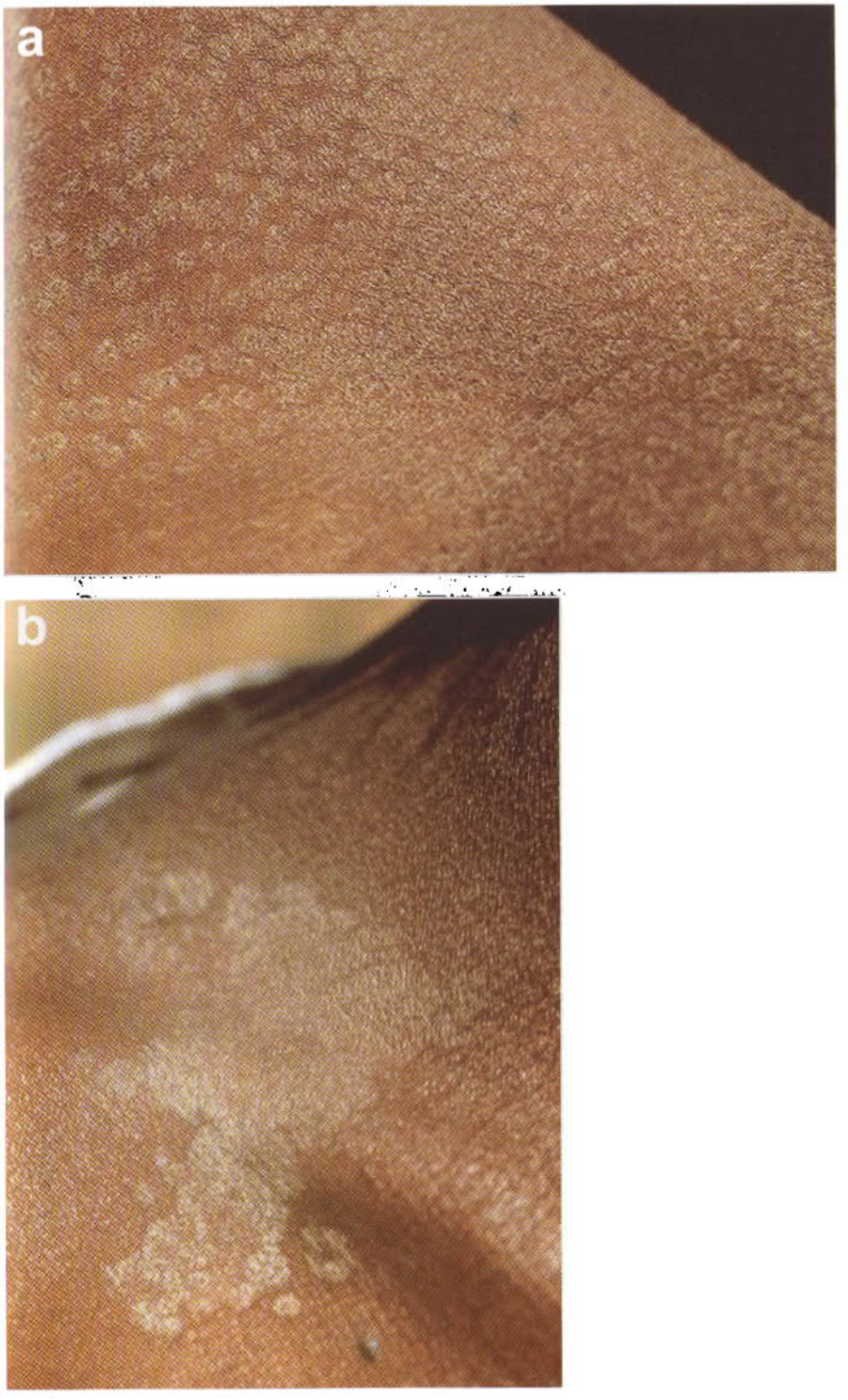
- Dimorphic yeast – *Malassezia furfur/Pityrosporum orbiculare*;
- Ubiquitous commensal lipophilic yeast.

Making the diagnosis

- Suspect on clinical examination;
- Must be distinguished from tinea corporis (*Trichophyton rubrum*, granular variant);
- Skin scraping or tape stripping with adhesive tape for direct microscopy is diagnostic – 'spaghetti and meatballs' or 'grapes and vines' pattern of clustered yeasts and pseudo-hyphae. Culture not usually required but can be done.

Significance

- Cosmetic but easily confused with tinea corporis.



a *Darwin sunburn on the back of the shoulder.*
b *White handkerchief pattern of pityriasis versicolor.*

Tinea Corporis

Synonym: Ringworm on the body.

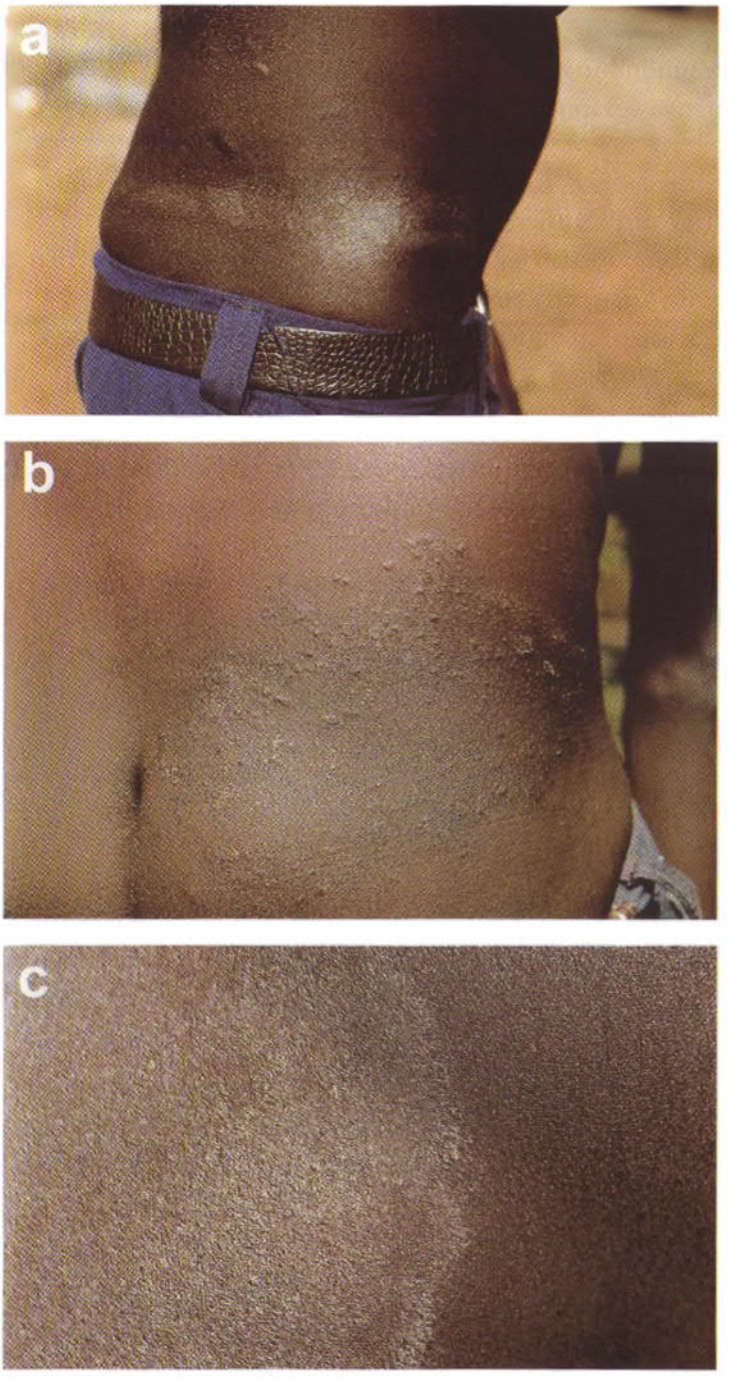
Summary: Asymmetrical superficial fungal infection of the trunk or limbs, which may become impetiginised (secondary bacterial infection).

CLINICAL DESCRIPTION

- Predominantly affects the trunk, sometimes limbs or groin (tinea cruris, jock itch), less commonly affects the face (tinea faciei);
- Usually asymmetrical – can be bilateral;
- Solitary lesion in 60% but can be multiple;
- Typically begins under the beltline
  – Initially feels sticky, earliest sign is scaling;
- Then appears above the beltline and/or extends down onto buttocks;
- Can be itchy particularly in sweaty areas;
- Signs include spreading edge, scaling, increased pigmentation and increased skin markings;
- May include thickened darker spots (papules) or be generally thickened with prominent skin markings and increased skin colour (lichenification);
- Scaling may be minimal or prominent;
- May present as circular patches with scale most prominent at the edge
  – Or most of the affected area may show scaling;
- Inflammation, blisters (vesicles), weeping, secondary infection – rare or absent in Aboriginal populations although in Caucasians is often inflammatory
  – Wood's light examination negative – no fluorescence;
- Nails may also be involved (10%);
- Tends to be chronic but may resolve spontaneously;
- Secondary bacterial infection is often underestimated.

Epidemiology

- Endemic in tropical regions;
- Children, adolescents and young adults are commonly affected;
- Usually spread person-to-person, less commonly indirectly from contaminated objects.



a *Typically* Trichophyton rubrum *infection begins under the beltline.*

b Trichophyton rubrum *spread can be both upwards from the beltline and downwards over the buttock. The eruption is asymmetrical with increased pigmentation, scaling and papules.*

c *Tinea corporis: A sharply defined, scaly spreading edge, but most of the affected area also shows some scaling.*

Causes

- Usually anthropophilic fungi
  - *Trichophyton rubrum* (granular variant) most common in northern Australia;
  - *Trichophyton violaceum* and *T. tonsurans* more commonly isolated on culture in central and southern Australia;
  - *Epidermophyton floccosum* can infect skin folds (e.g. groin (tinea cruris)) but usually begins between toes (tinea pedis).
- *Trichophyton concentricum* (tinea imbricata) – rare or absent in Australia.

Making the diagnosis

- Clinically suggestive;
- Adhesive tape can be applied to scaly skin, stripped off, and then examined microscopically for hyphae to distinguish from pityriasis versicolor;
- Skin scrapings for microscopy and fungal culture – specimens can be spread on culture medium in the field or will remain viable up to 30 days to be sent to a distant laboratory;
- Skin scrapings to obtain sufficient scale can be difficult if scaling is minimal;
- *Trichophyton rubrum* (granular variant) scale is typically very adherent and difficult to scrape, resulting in superficial bleeding.

Significance

- High prevalence of anthropophilic infection in Aboriginal populations relates to overcrowding and poor living conditions.

Scabies

Synonym: 'The itch', itch mite.

Summary: Infestation by a mite that burrows into the superficial skin and excites an allergic generalised itch and variable rash, which commonly becomes secondarily infected (impetiginised). Crusted scabies is a less common form in which itch may be absent.

CLINICAL DESCRIPTION

- Typical sites of burrows include between the fingers and toes (interdigital), wrist (ventral, flexor aspect), armpit (axillary) folds, nipples, elbows and penis;
- Rash is variable and may consist of excoriations (scratch marks), or appear to be eczematous or papulo-nodular;
- In infants it is often vesicular involving the scalp, palms and soles;
- Itch is prominent;
- Secondary bacterial infection (impetiginisation) is common and is estimated to be the underlying cause of 50–70% of all streptococcal impetigo.
- Crusted scabies (Norwegian scabies), an unusual variant, appears as an extensive white scale and is diffuse or patchy, including under fingernails and toenails, palms and soles (hyperkeratosis). (Also see page 126).
- Usually not itchy.

Epidemiology

- Endemic and in some places epidemic – up to 50% of children and 25% of adults infested in some remote communities;
- Spread person-to-person;
- All ages, both sexes;
- Multiple overlapping epidemic cycles.

Causes

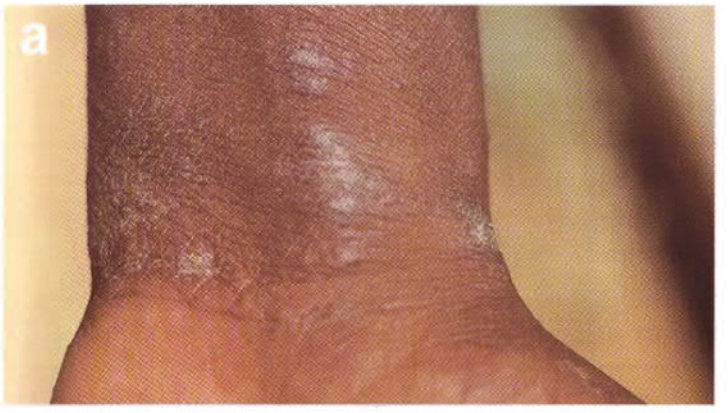
- *Sarcoptes scabiei* var. *hominis* (human mite)
  - Despite close proximity of dogs, not due to dog scabies (scabietic mange, *Sarcoptes scabiei* var. *canis*);
- Generalised itch and rash are an allergic response to the infestation;
- Crusted scabies due to an abnormal host immune response or a failure to scratch.

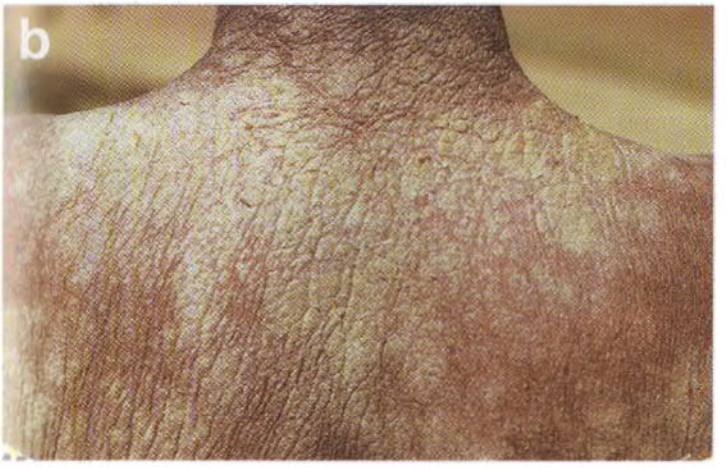
Making the diagnosis

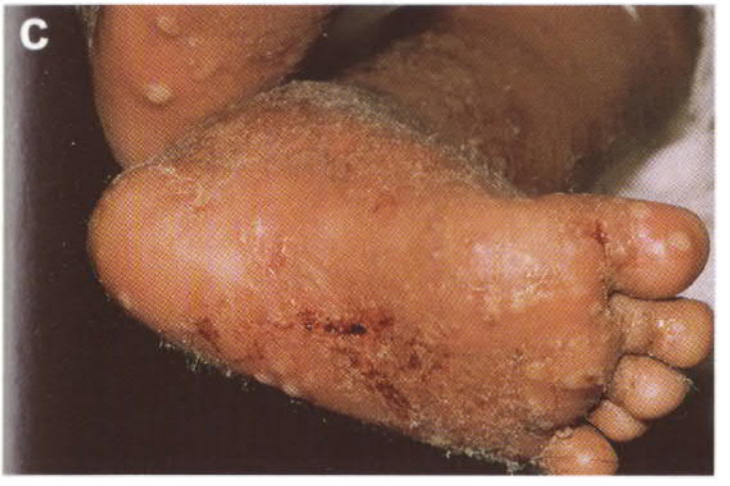
- Suspect clinically;
- Skin scrapings from burrows between fingers and from wrist for direct microscopy to demonstrate the adult mite, eggs (ova) and faecal pellets. In classic scabies there are few mites (see page 25);
- The adult mite can sometimes be extracted from a vesicle at the tip of a burrow clinging to the end of a needle;
- In crusted scabies, numerous adult mites are seen in scrapings taken from any scaly area. This may need to be distinguished from psoriasis although this is apparently absent or rare in Aborigines.

Significance

- Endemic scabies is a marker of overcrowding and poor living conditions;
- Secondary impetiginisation is very common;
- Crusted scabies is a sign of an abnormal host response including HTLV-1 infection, immunosuppression, neuropathy or paralysis, arthropathy, dementia or other mental disturbances.







a *Scaly lesions on flexor aspect of wrist in classic scabies.*

b *Diffuse white scale of crusted scabies over the upper back.*

c *Scabies involving the sole of the foot in an infant. (See also Figure (b) (page 87) Secondary impetiginisation of scabies on the wrist.)*

Impetigo

Synonym: Pyoderma, skin sores.

Summary: Impetigo is a primary contagious bacterial infection of the skin usually caused by group A streptococci, which may be followed by acute post-streptococcal glomerulonephritis; Impetiginisation is the secondary infection of skin already damaged by other infections or infestations, insect bites or other forms of skin trauma.

CLINICAL DESCRIPTION

- May present as blisters (bullae), weeping sores (erosions), crusted (typically honey-coloured) or scaly areas of skin;
- Smaller, similar but more recent spots close by (i.e. satellite lesions);
- May be localised or extensive, any site;
- Impetiginisation is secondary to underlying skin infestations/ infections such as scabies, lice, tinea or skin trauma including burns, bites and stings.

Epidemiology

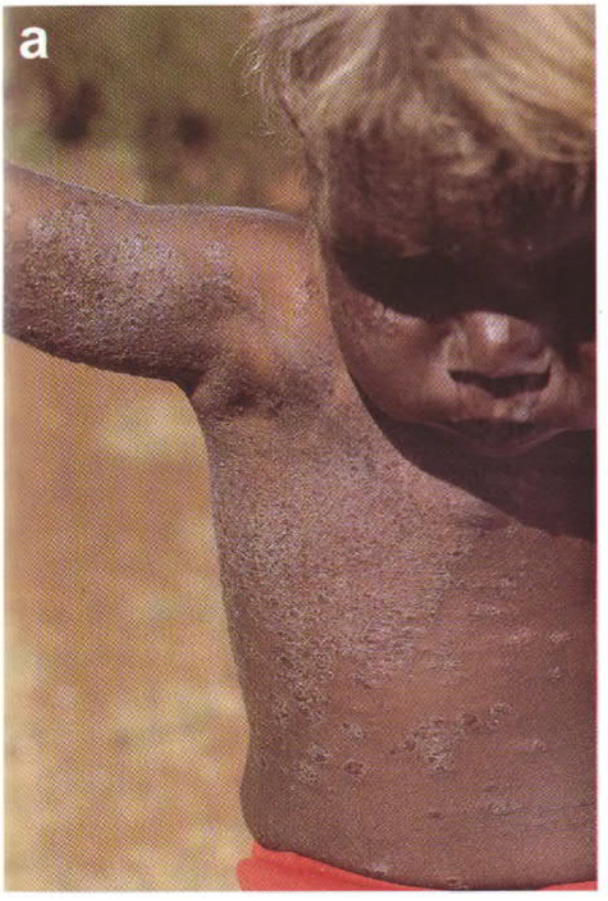
- Point prevalence in central and northern Australia – 10–70% of Aboriginal children in remote communities;
- Overcrowding, poor hygiene and pre-existing skin diseases predispose to this infection;
- Common in remote and urban communities.

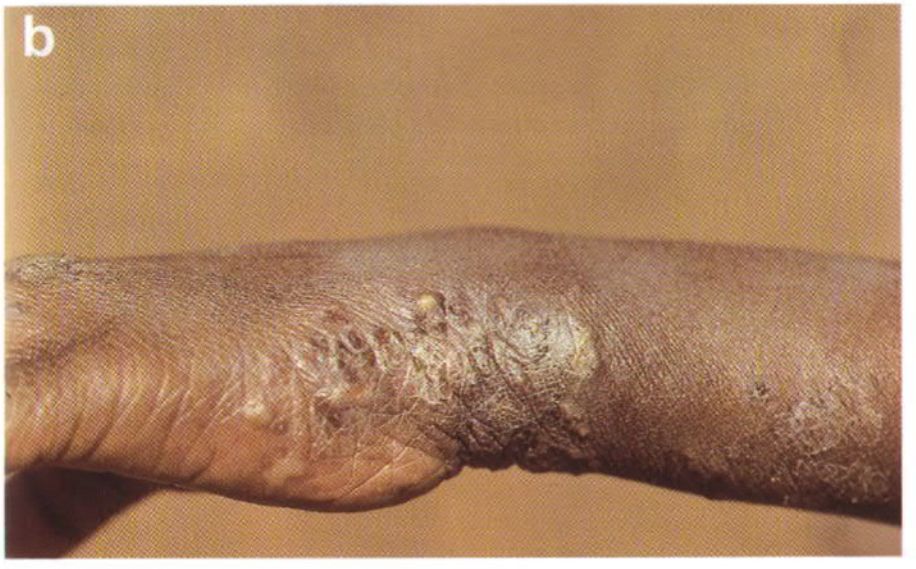
Causes

- *Streptococcus pyogenes* (group A streptococci, beta haemolytic strep.)
  - Usual primary pathogen – 80%, particularly in the tropics;
  - Enormous diversity of isolates;
  - Scabies commonly underlies invasive infection;
  - Important cause of acute post-streptococcal glomerulonephritis;
  - May be linked to acute rheumatic fever.
- *Staphylococcus aureus* usually a secondary wound coloniser.

Making the diagnosis

- Skin swabs for bacteriology – microscopy and culture;
- Important to examine again after treatment of the bacterial infection for a possible underlying predisposing problem.





a *Extensive scaly impetigo over the arm and trunk.*

b *Secondary impetiginisation of scabies on wrist.*

Significance

- Group A streptococcal skin infection may result in post-streptococcal glomerulonephritis and chronic end-stage renal failure.

Kava Dermopathy

Synonym: Crocodile skin.

Summary: Generalised scaly eruption with localised thickened areas resulting from heavy kava consumption, which can be associated with neuropathy.

CLINICAL DESCRIPTION

- Generalised scaly appearance;
- White scruff or scale;
- Starts on the head, face and neck;
- Extends over the body and progressing eventually to the feet;
- Thickened scaly (keratotic) plaques.

Epidemiology

- Since the uptake of kava drinking by Aboriginal communities in tropical regions;
- From the Pacific Islands (Polynesia, Melanesia).

Causes

- Heavy kava consumption;
- Precise chemical cause unknown.

Making the diagnosis

- History;
- Resolves promptly when kava drinking is ceased;
- Associated features include emaciation and neuropathy.

Significance

- Neuropathy.

Normal Variations in Pigmentation

CLINICAL DESCRIPTION

1 Hypopigmentation

Lighter skin colour but not white

- Bilateral, symmetrical macules over the upper cheeks (malar) and around the nose (ala nasi, perinasal);
- Hypopigmented naevi/birthmarks.

2 Depigmentation

Total loss of pigment so the skin is white

- Halo naevus – central melanocytic naevus with a white surrounding halo usually seen in adolescents.

3 Hyperpigmentation

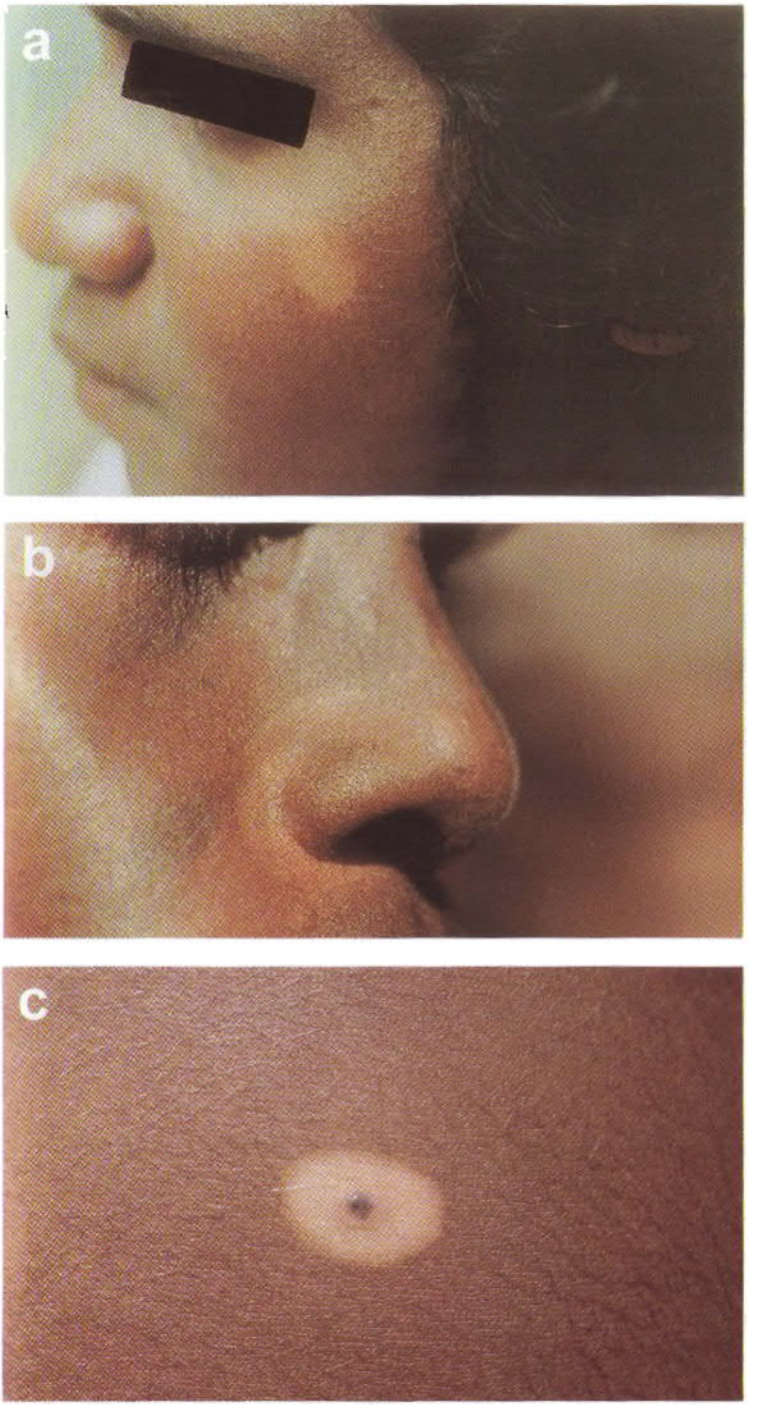
Increased skin colour

- Moles (melanocytic naevi) – junctional, compound, intradermal, blue;
- Mongolian spot (also see page 92);
- Solar hyperpigmentation - areas exposed chronically to sun (i.e. face, neck and hands) tend to be darker than areas usually covered by clothing.

Also see page 4.

Making the diagnosis

- **To distinguish normal hypopigmentation from leprosy – bilateral and symmetrical, no associated features such as changes in sensation or reduced sweating.**



a *Bilateral hypopigmentation on malar regions.*

b *Bilateral hypopigmentation around the nose.*

c *Halo naevus on the trunk. To distinguish from vitiligo (which is rare but has been documented in Aboriginal Australians) note the central melanocytic naevus.*

Mongolian Spot

Summary: Almost universal at birth presenting as a purplish discolouration usually over the buttocks, which does not change in colour but is gradually obscured by the normal darkening of the skin with age.

CLINICAL DESCRIPTION

- Present at birth;
- Grey/blue/purple discolouration but the intensity varies between individuals;
- Flat and not palpable (macular);
- Round or oval in shape;
- Clearly defined;
- The skin is otherwise normal;
- Size varies from less than 1 cm up to covering the entire buttocks;
- Most commonly over the lower back (lumbosacral) and buttock areas;
- Occasionally elsewhere on the back or upper or lower limbs;
- May be solitary or multiple (up to ten or more);
- Initially may become darker in colour for some weeks or months after birth;
- Become increasingly difficult to see as the skin darkens with age so usually no longer visible by 12–18 months of age, but in some may remain apparent up to 13 years of age.

Epidemiology

- Very common in all dark-skinned groups;
- Both sexes.

Causes

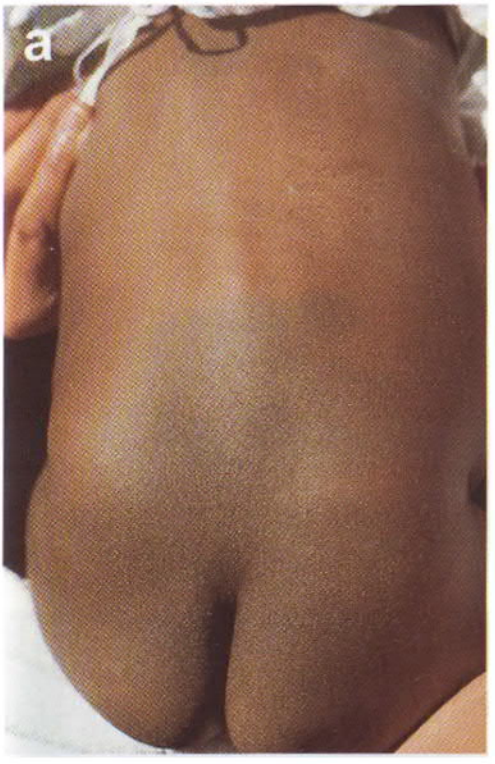
- Arrest of melanocytes in dermis during embryonal migration from neural crest;
- Genetic tendency.

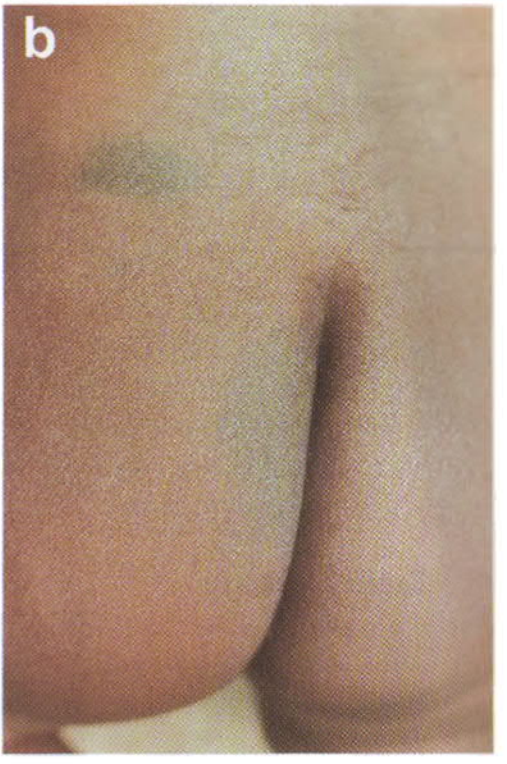
Making the diagnosis

- To distinguish from bruises – the colour of a Mongolian spot does not evolve from purple through yellow before fading;
- Skin biopsy for histology to demonstrate the slender elongated melanocytes in the lower half of the dermis.

Significance

- None but not to be confused with child abuse.





a *Extensive Mongolian spot over the sacral and buttock area.*
b *Multiple Mongolian spots over the lower back and buttock.*

Pseudo-Acanthosis Nigricans

Summary: Darker thickened skin in the folds, particularly of the armpits and neck, associated with obesity, to be distinguished from true acanthosis nigricans, which may indicate systemic disease particularly diabetes mellitus.

CLINICAL DESCRIPTION

- Involves skin folds (flexures) – particularly nape of neck, armpits;
- Increased pigmentation/darker skin;
- Velvety texture/thickened skin;
- Multiple skin tags often also present;
- Associated with obesity.

Epidemiology

- Common in dark-skinned groups;
- From adolescence onwards;
- Both sexes.

Causes

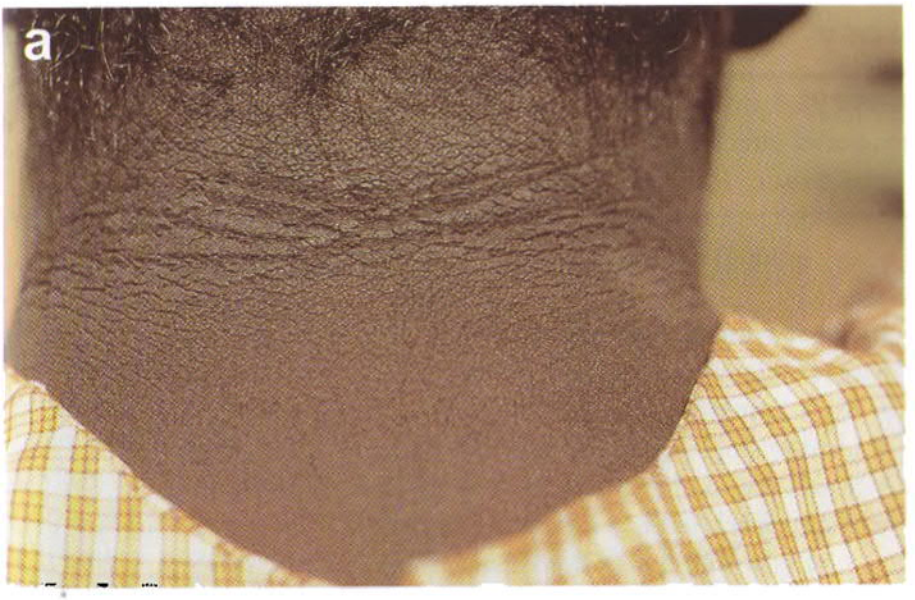
- Normal reaction in pigmented skin to the rubbing together of opposing skin surfaces when combined with heat, sweat and friction in hot climates;
- Obesity may induce insulin-resistance.

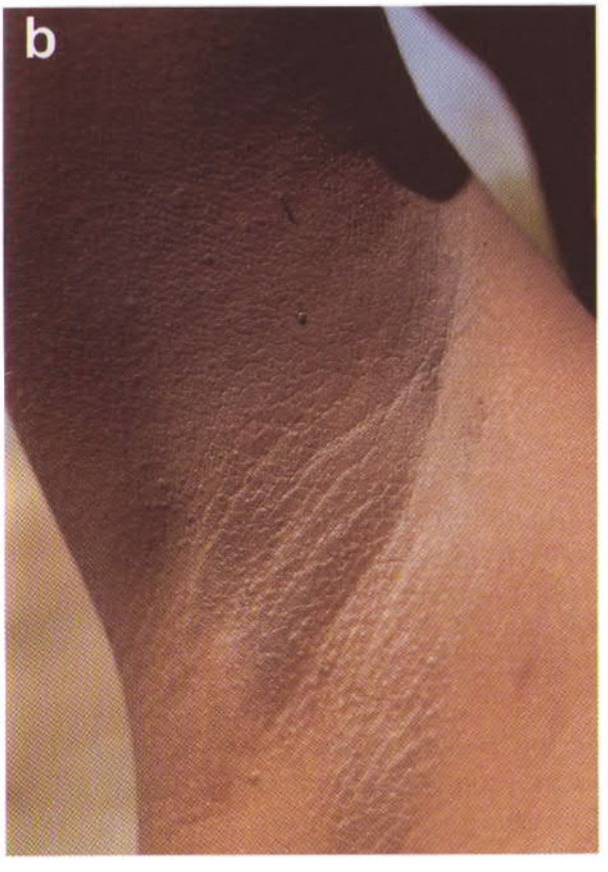
Making the diagnosis

- To distinguish from true acanthosis nigricans – good health, obesity and absence of associated features suggests pseudo-acanthosis nigricans;
- Acanthosis nigricans
  Familial – onset in infancy or childhood, female predominance, not associated with obesity, usually mild, worse at puberty and then stable or regresses;
  Endocrinopathies – e.g. diabetes mellitus, and others;
  Medications – corticosteroids, oestrogens,and others;
  Syndromal – very rare, associated features of the syndrome;
  Malignant – usually itchy and severe including mucous membranes.

Significance

- Secondary to obesity and its attendant health problems;
- Not to be confused with true acanthosis nigricans.





a *Velvety hyperpigmentation of the nape of neck.*
b *Pseudo-acanthosis nigricans in the axilla.*

Leprosy

Summary: A disorder of skin and nerves due either to ongoing mycobacterial infection or the host response to this, which can result in peripheral sensory loss and physical and social disabilities.

CLINICAL DESCRIPTION

1 Indeterminate leprosy

- Hypopigmented coppery patches;
- Most commonly on the face, but can occur anywhere;
- Usually single or few in number – asymmetrical;
- Small, round, flat (macular) – never thickened or raised;
- Slight decrease in sweating and sensation;
  – May be difficult to demonstrate, especially in children.

2 Tuberculoid leprosy

- Hypopigmented coppery patches (not white);
- Usually solitary or few in number – asymmetrical;
- Can occur anywhere;
- When on the back the patch seldom crosses the midline except if sacral;
- Irregular, slightly raised, fairly well-demarcated edge (plaque);
- Non-sweating (anhydrotic) with reduced sensation;
- Loss of hair centrally;
- May repigment centrally.

3 Lepromatous leprosy

- Numerous widespread lesions with little or no change in pigmentation;
- Symmetrical.

4 Borderline leprosy

- Various clinical presentations depending upon organism numbers and host response but does not affect pigmentation.

Epidemiology

- Mostly acquired in tropics and subtropics;
- Endemic in northern areas – 7–15 new cases are reported each year Australia-wide;
- Route of infection unknown;
- Low infectivity (or perhaps subclinical infection) even with prolonged close proximity – only 5% of marriage partners acquire disease and few documented cases in medical attendants or other carers.

Causes

Mycobacterium leprae

- Genetic predisposition to develop clinical disease;
- Variable immunological response
  - Indeterminate leprosy – an early transitory stage, immunological state not yet determined, organisms rarely detected;
  - Tuberculoid leprosy – an excessive immune response causes the symptoms and signs, no organisms present;
  - Lepromatous leprosy – an absent host response permits mycobacteria to proliferate, numerous organisms;
  - Borderline leprosy – immunologically unstable, organism numbers range from none to numerous.

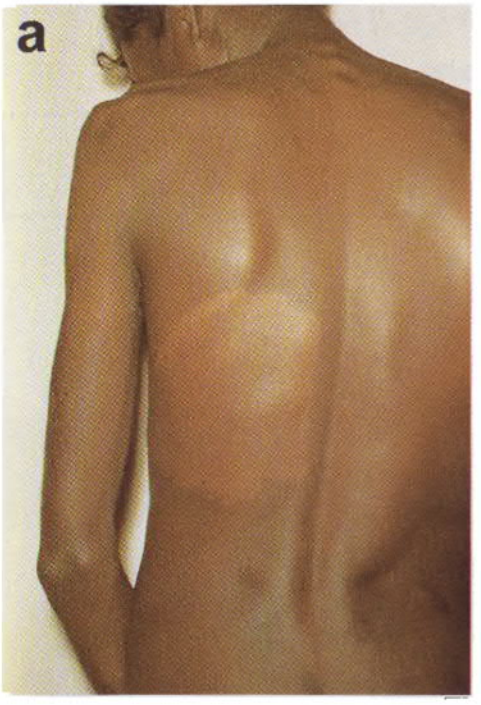
Making the diagnosis

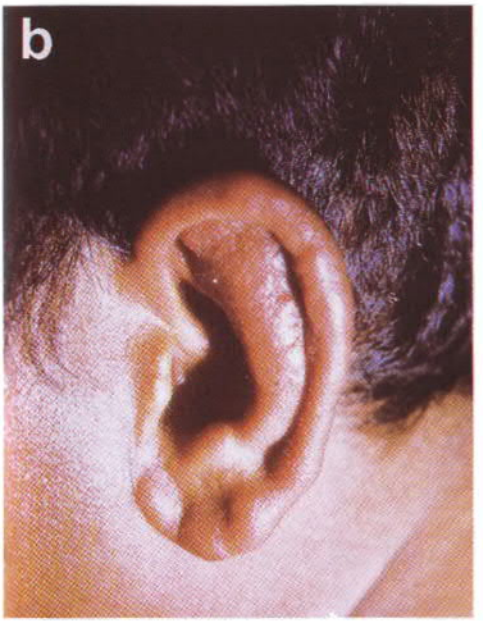
- Important to investigate thoroughly;
- Clinical examination for other features including:
  - Indeterminate leprosy – acquired ichthyosis (see page 76);
  - Tuberculoid leprosy – palpable peripheral nerves, secondary changes due to anaesthesia (see pages 105 and 129);
  - Lepromatous leprosy – skin signs include macules, papules and nodules, succulent earlobe (or residual wrinkling), thinning and then loss of eyebrows and eyelashes (madarosis; see page 62), leonine facies (see page 30), dry skin (secondary/acquired ichthyosis; see page 76) on the legs, neuropathic ulcers and callosities (see pages 126 and 128), mucosal papules/nodules may lead to nasal and palatal perforation;
  - Borderline leprosy – skin signs depend upon the degree of host response and organism numbers, may include annular or bizarre-shaped macules or punched out plaques, dry non-sweating (anhydrotic) skin or changes similar to lepromatous leprosy.
- Dermal scrapes for acid-fast bacilli (AFB) (also see methods section, page 26) – positive in lepromatous leprosy, negative in tuberculoid;
- Skin biopsy including special stains for AFB;

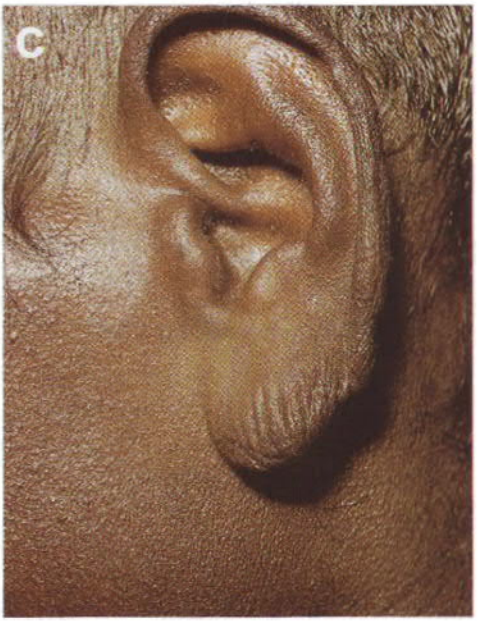
- Nerve conduction studies;
- Exclude differential diagnoses including pityriasis versicolor and tinea by Wood's light examination (leprosy – pigment change not clearly shown) and skin scraping for fungal microscopy and culture and/or trial of antifungal therapy. Yaws has now been apparently eradicated from Australia, but secondary yaws may present as slightly scaly hypopigmented patches.
- Organism does not grow *in vitro* so culture is not helpful.

Significance

- A disease of antiquity carrying unwarranted social stigma and a potential for considerable physical disability that is secondary to nerve damage.







a *Tuberculoid leprosy of the mid back – coppery hypopigmented patch restricted to one side, not crossing the midline.*

b *Succulent earlobe of lepromatous leprosy.*

c *Residual wrinkling remaining after resolution of the succulent earlobe.*

(See also Folded forehead (page 129); Madarosis (page 31); Neuropathic ulcer (page 63).

Post-Inflammatory and Post-Traumatic Hyper- or Depigmentation

Summary: Change in skin colour following skin inflammation or trauma.

Significance

- Cosmetic.

DEPIGMENTATION

CLINICAL DESCRIPTION

- Depigmentation – total loss of pigment;
- May follow accidental or deliberate skin trauma
  – Including nice marks and sorry cuts (see page 104);
- May follow inflammatory dermatoses
  Including discoid lupus erythematosus (see page 34) and shingles (herpes zoster);
- Can be permanent;
- May be associated with keloid scarring.

Epidemiology

- Widespread and common in dark-skinned races.

Cause

- Loss of melanocytes following inflammation involving the basal layer of the epidermis where the melanocytes are located.

Making the diagnosis

- Distinguish from vitiligo (which has been documented in Aborigines, although rare) – on history, no associated scarring with vitiligo.

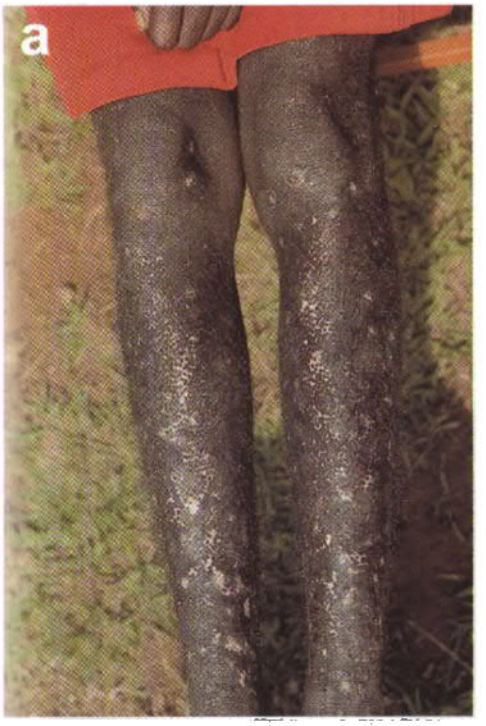
HYPERPIGMENTATION

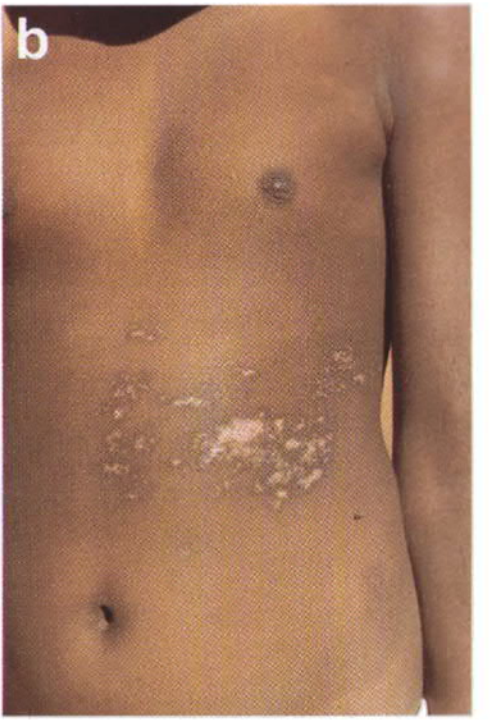
CLINICAL DESCRIPTION

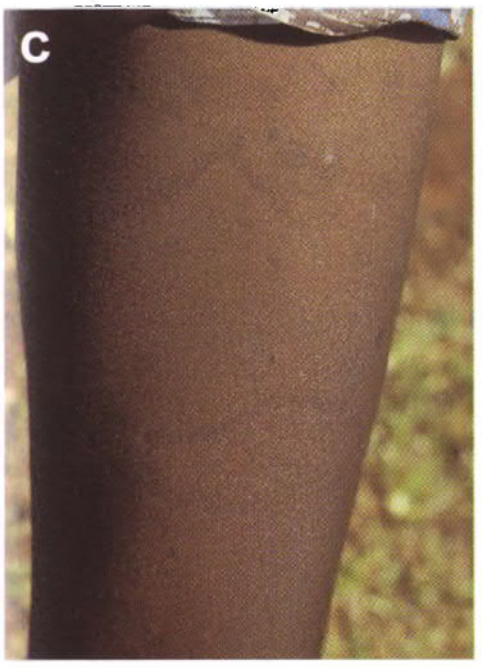
- Increased pigmentation at sites of skin trauma or inflammation.

Causes

- Melanin incontinence – loss of melanin from epidermal cells into the dermis, where it is taken up by macrophages (melanophages) similar to a tattoo;
- Typically follows lichen planus.







a *Depigmentation following injuries while hunting mud crabs in mangroves – 'leopard skin'.*
b *Changes in skin colour following shingles.*
c *Hyperpigmented scars from a jellyfish sting.*

Lateral Malleolar Bursitis

Synonym: Gambler's ankle.

Summary: Prolonged pressure from sitting cross-legged. Can initially cause thickening and then ulceration over the lateral malleolus of the ankle.

CLINICAL DESCRIPTION

- Initially scaly thickening (hyperkeratosis) over the lateral malleolus (outer aspect of the ankle) and base of the fifth metatarsal (base of little toe);
- Skin and lateral wall of the bursa ulcerate;
- A probe can be inserted some distance into the bursa.

Epidemiology

- Both sexes but a male predominance.

Cause

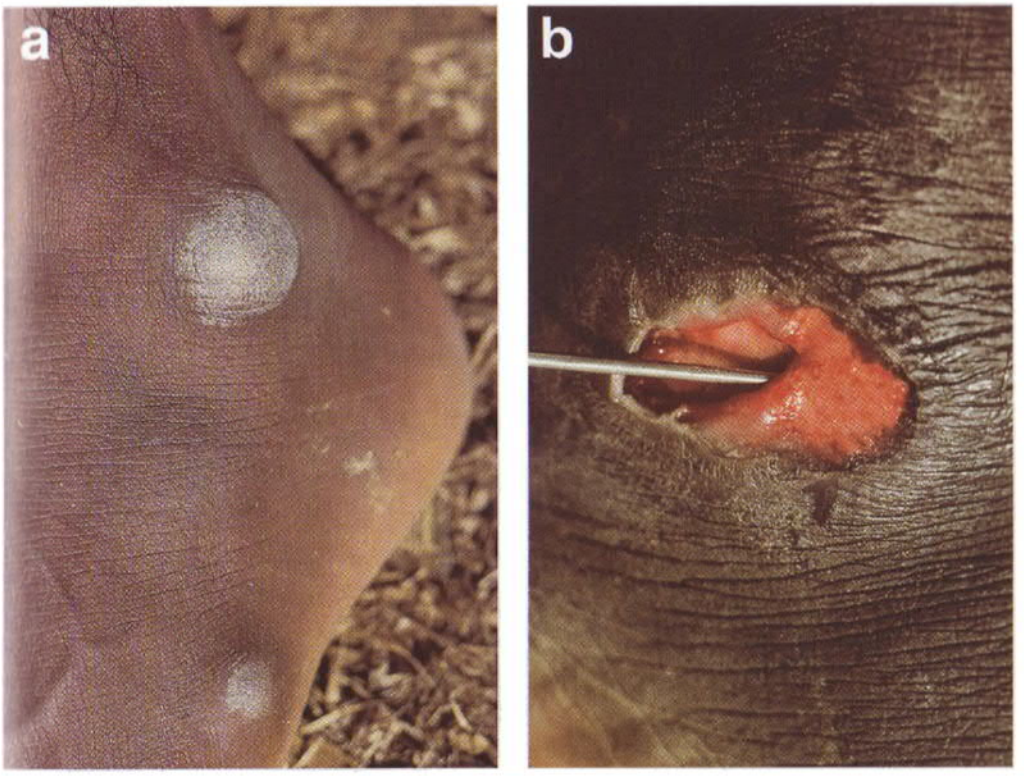
- Pressure from sitting cross-legged on the hard ground for long periods of time.

Making the diagnosis

- Usually clinical;
- Need to exclude neuropathic ulcers (e.g. leprosy, diabetes mellitus) and vascular disease.

Significance

- Benign unless complicated by infection;
- Cosmetic concerns may arise.
- May be a sign of systemic disease.



a *Hyperkeratosis and thickening over the lateral malleolus and base of fifth metatarsal.*
b *Probe inserted into ulcerated lateral malleolar bursa. (Reprinted with permission from* Global Dermatology. *LC Parish, LE Millikan (eds). Springer-Verlag, New York, 1994.)*

Traumatic Ulcers and Sores

Summary: Sores and ulcers following accidental or deliberate skin damage or secondary to nerve damage causing anaesthesia of the skin may become secondarily infected and heal with altered pigmentation or keloid scarring.

CLINICAL DESCRIPTION

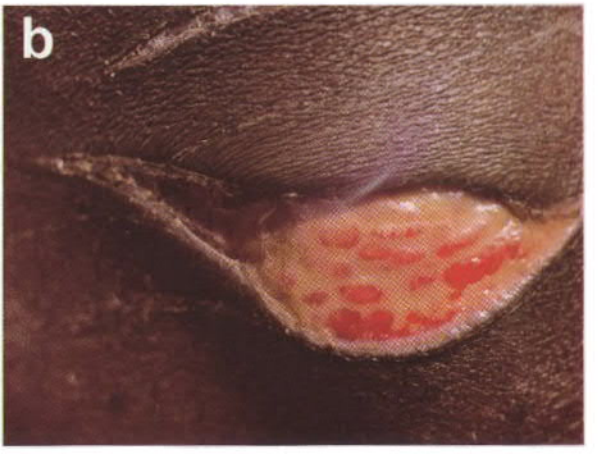
- Loss of full-thickness epidermis with or without dermal loss, whether partial or total;
- Commonly become secondarily infected;
- Linear or patterned nature may give a clue to diagnosis.

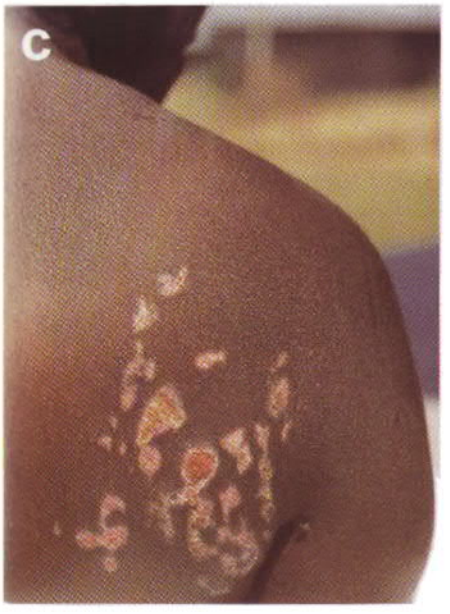
1 Accidental

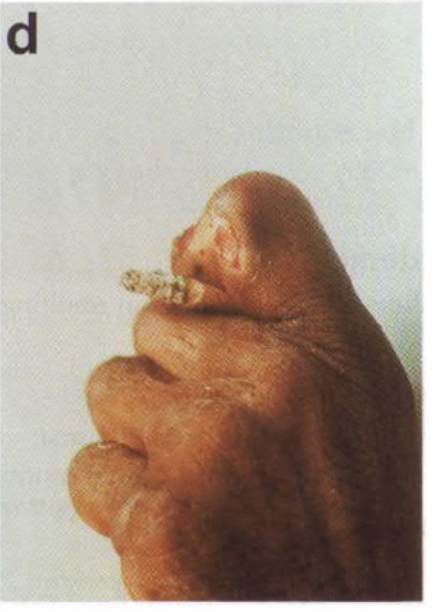
- Fauna and flora (e.g. dogs, stingrays, plants with thorns, sharp-edged leaves);
- Burns are common due to lifestyle;
- Injury from domestic and external disputes is common among Aborigines, both males and females;
  - The injuries are as diverse as the weapons;
  - Include abrasions and lacerations.

2 Deliberate

- Sorry cuts
  - Self-inflicted wounds made at the time of the death or burial of a relative or close friend
  - Men usually use a knife to slash the outer aspects of the arms and thighs;
  - Women usually use a stone, wooden club (nulla nulla) or heavy stick to pound the scalp (see page 65);
  - Young girls may lacerate their hips and calves when a brother is being ritually circumcised as a sign of sharing pain.
- Nice marks
  - Prepubertal and adolescent girls in central Australia
  - Burns on the back of the hands and forearms;
  - Made using glowing twigs or burning cigarettes.
- Ceremonial rituals
  - Nasal septum (nose holes) and earlobe piercing
  - Touching the back of the shoulders with a branch of burning leaves.







a *Penetrating wound from a stingray 2 weeks earlier.*

b *Recent sorry cut on postero-lateral thigh.*

c *Ceremonial burns over the right shoulder – not shingles. In some regions a ritual tribal dance involves the touching of shoulders with a branch of burning leaves.*

d *Damage resulting from a cigarette burn in tuberculoid leprosy. (Reprinted with permission from Dr J Hargrave).*

- Traditional treatments
  - scarification
  - numerous incisions made into the skin (e.g. of the forehead for headache, upper back for bronchitis, lower back for kidney pain or backache);
  - Cautery – a glowing piece of burning wood called a fire-stick is often used to cauterise lesions (e.g. primary syphilis or donovanosis).
- Punishment ('payback') – burns, spear wounds, knife wounds
  - Inflicted as punishment for transgressors of tribal laws;
  - Vary from slight to severe or fatal, may result in permanent disability.
- Self-mutilation – depressive or other mental illness or suicide attempts.

3 Neuropathic

- May complicate neuropathy (e.g. leprosy, diabetes mellitus).

Epidemiology

- Widespread and common resulting from environmental hazards and rituals.

Causes

- Accidental or deliberate trauma;
- Loss of sensation makes skin more prone to unrecognised trauma.

Making the diagnosis

- Usually on history;
- May need to distinguish from keloid scars, which can themselves ulcerate.

Significance

- Depends on severity and cause.

Ulcers and Sores due to Infections

ECTHYMA

Summary: Common bacterial skin infection associated with poor socio-economic conditions in which a deep ulcer is hidden by an adherent crust.

CLINICAL DESCRIPTION

- Begin as small blisters or pustules;
- Adherent crust forms – difficult to remove;
- Deep irregularly shaped ulcer beneath the crust;
- Painful;
- Buttocks, thighs and legs;
- Increases in size by direct extension;
- Multiple lesions due to auto-inoculation;
- May follow insect bites and stings;
- Chronic;
- Heals with scarring.

Epidemiology

- Very common, especially in tropics;
- Can affect any age group.

Causes

- Group A streptococcus (*Streptococcus pyogenes*) – Gram-positive coccal bacterium;
- Commonly mixed infection of *S. pyogenes* and *Staphylococcus aureus*.

Making the diagnosis

- Ulcer seen after removal of the crust;
- Skin swabs from ulcer for bacteriology – microscopy and culture.

Significance

- Associated with poor hygiene, malnutrition and minor skin trauma.

BOILS

Synonym: Furuncles.

Summary: Most commonly seen in hairy areas of adult males. This painful lump is caused by *Staphylococcus aureus* ulcerates, and discharges then heals with scarring.

CLINICAL DESCRIPTION

- Deep infection of hair follicle;
- Occur on hairy skin subject to friction (i.e. back of neck, scalp, armpit, breast, thigh, buttocks);
- May be single or multiple in crops;
- Begins as a small lump based on a hair follicle (follicular nodule), becomes pustular and then necrotic to form a sore (ulcer);
- Painful;
- Discharge – blood-stained pus;
- Heals with scarring.

Epidemiology

- Most commonly affects adult males.

Cause

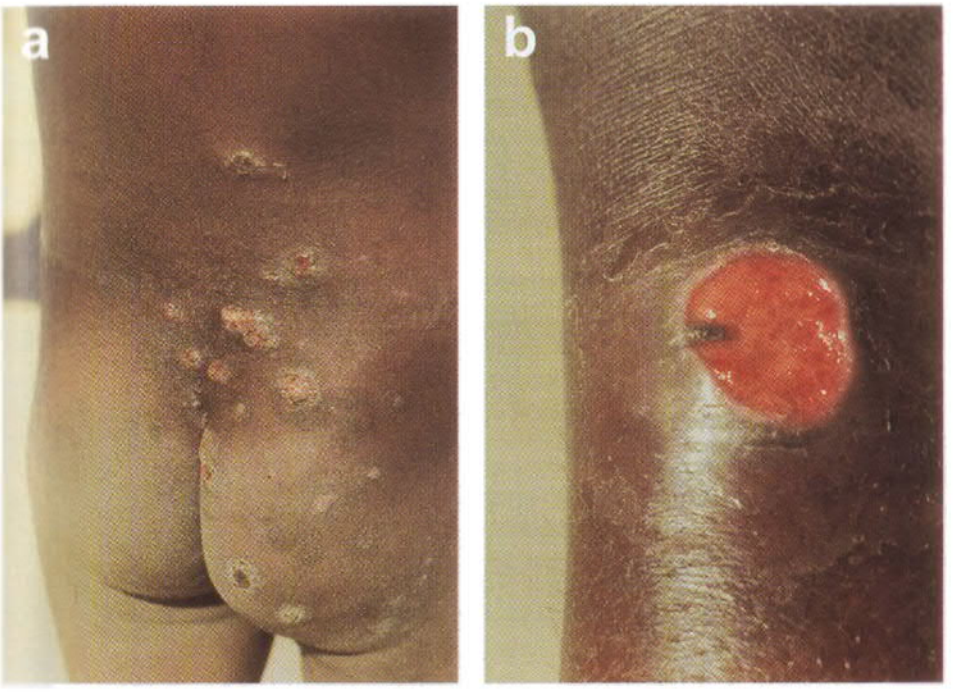
- *Staphylococcus aureus* – Gram-positive coccal bacterium, normal skin flora.

Making the diagnosis

- Usually clinical;
- Skin swab for bacteriology – microscopy and culture.

Significance

- Associated with malnutrition, anaemia, diabetes mellitus;
- A cause of significant morbidity and health costs.



a *Ecthymatous sores over the buttocks of a child.*
b *An ulcer resulting from a boil.*

Keloid and Hypertrophic Scarring

Synonym: Cicatrisation.

Summary: Thickened scars commonly follow any form of skin damage in dark-skinned individuals and can result in significant physical disability if the scar crosses a joint.

CLINICAL DESCRIPTION

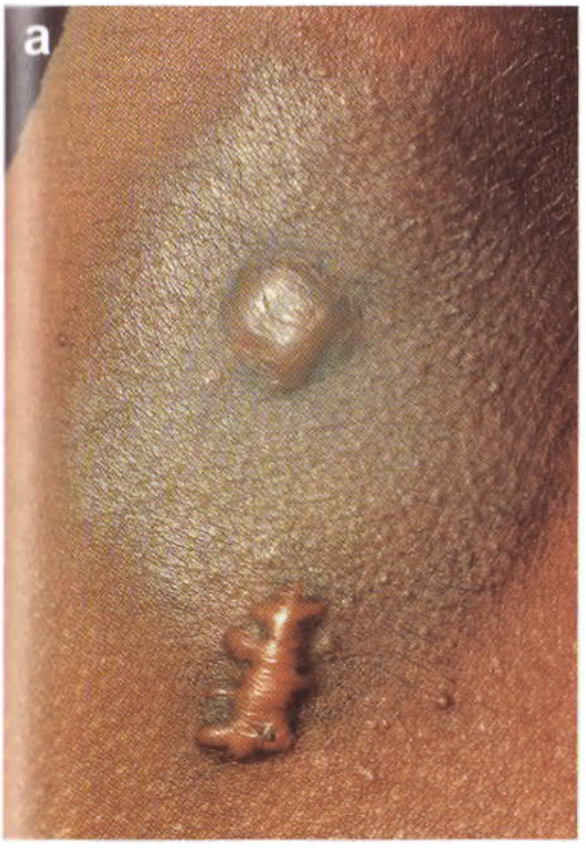
- Commonly follow any form of skin trauma.
- Keloid is an overgrowth of scar tissue in and around the site of injury
  - Firm to hard, raised, smooth, often shiny, usually skin-coloured;
  - May be painful, tender or itchy;
  - Range in size from a few millimetres up to and over 20 cm;
  - May involve any site but most commonly occur on the back, upper chest (especially presternal), over the outer surface of the upper arms, face, neck and earlobes;
  - In flexures, keloids can fix the joints (immobile) resulting in disability.
- Hypertrophic scar is an overgrowth of scar tissue that remains within the site of an injury;
- May follow – tribal/ritual scars - following the ritual incisions over the upper abdominal wall and chest, ash and ochres rubbed into the cuts encourage the development of thick raised scars;
  - Some tribal ceremonies traditionally required human blood obtained by cutting into the veins of the forearms resulting in linear scars or small, firm fibrous lumps (papules) overlying the course of a vein(s);
  - Sorry cuts, punishments;
  - Injections, skin biopsies, surgical procedures, venesection, lacerations, ear piercing, burns;
  - Discoid lupus erythematosus, acne.
- May be complicated by flexion deformity if across a joint, or ulceration.

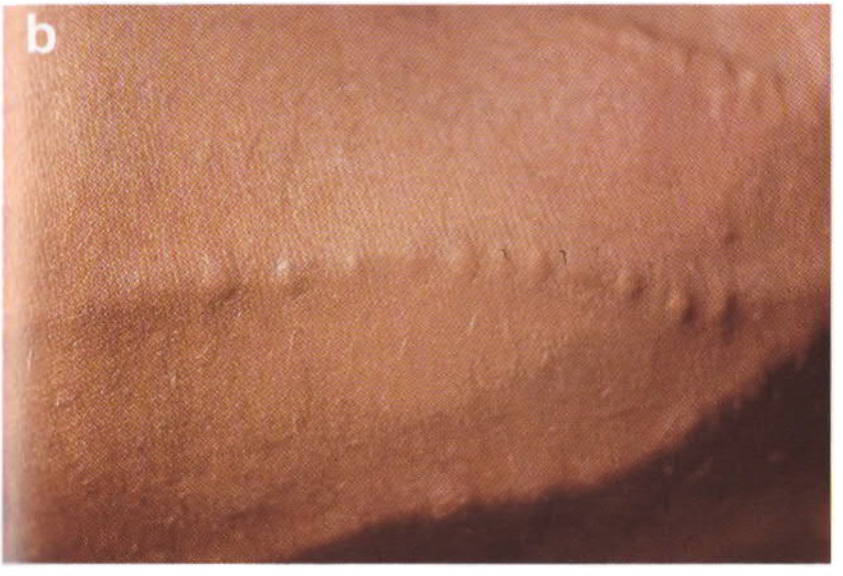
Epidemiology

- Common in the Aboriginal population and other dark-skinned groups.

Causes

- Genetic predisposition;
- Any skin trauma.





a *Keloid on the breast (adjacent to the areola and nipple) following an incisional biopsy.*

b *Fibrous nodules and linear scars on the forearm resulting from venesection for ceremonial rituals. History will distinguish this from sporotrichoid spread of some infections.*

Making the diagnosis

- Usually on history and clinical examination.

Significance

- Used to advantage for personal adornment but may be of cosmetic concern such as following a skin biopsy;
- Where a keloid scar crosses a joint, a fixed flexion contracture may result causing significant physical disability.

Effects of Fauna and Flora

Summary: A number of insects and plants can cause a variety of skin reactions.

CLINICAL DESCRIPTION

1 Arthropod bites (with or without venom)
- Include biting midges (sandflies), mosquitos, ticks, fleas, mites, black flies, march flies, leeches, ants.

2 Thorns, spikes, hairs, spines and bristles (with or without venom)
- Include bindi-eye sores (bindii dermatitis, bindy ii sores, Jo Jo dermatitis), caterpillar hairs (which may be carried considerable distances by wind), coral dermatitis, many fish, bristle-worms, starfish/sea urchins, prickly pear, plants with damaging hairs (e.g. *Hibiscus spp, Malachra fasciata, Cionachne cyathapoda*).

3 Contact irritants
- Include blistering beetles (acid beetles), marine sponges and plant juices (e.g. mangroves).

4 Parasite infestation
- Schistome dermatitis (bather's itch) resulting from *Austrobilharzia terrigaleusis* in lagoons on the New South Wales coast and in weedy stretches of rivers in inland Australia.

- Effects can include:
  - Single or multiple lesions;
  - Reaction may be due to sensitisation to previous exposure;
  - May leave foreign material in wound leading to foreign body granulomas;
  - May introduce infections – acute or chronic;
  - Effects may be chemical – irritant or allergic, urticarial, vesiculobullous.

Epidemiology

- Native and introduced species of fauna and flora;
- Vary by region, season, time of day, terrain and other ecological factors;
- Important to know the fauna and flora of the region and the patient's activities;
- Many Aboriginal people use bush medicines.

Cause

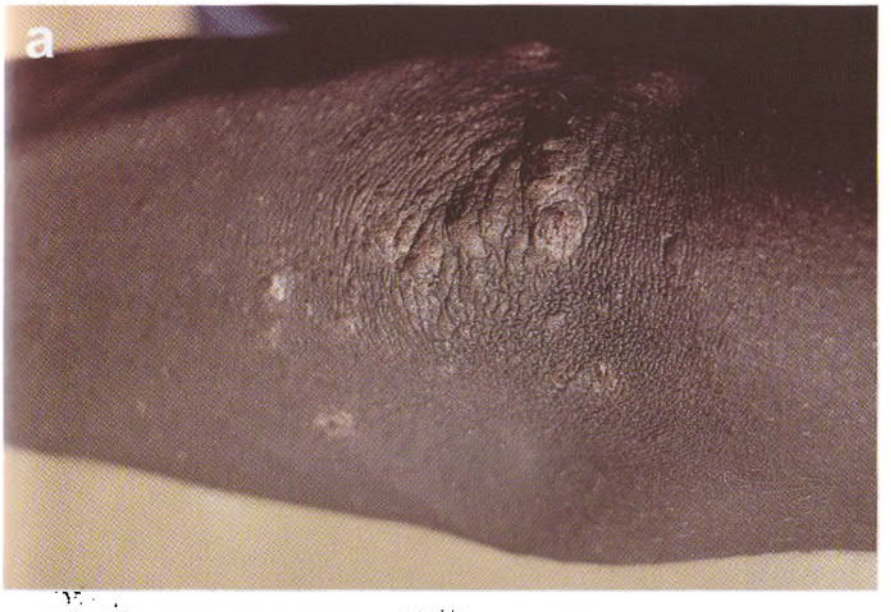
- Mechanical, chemical, immunological, infective.

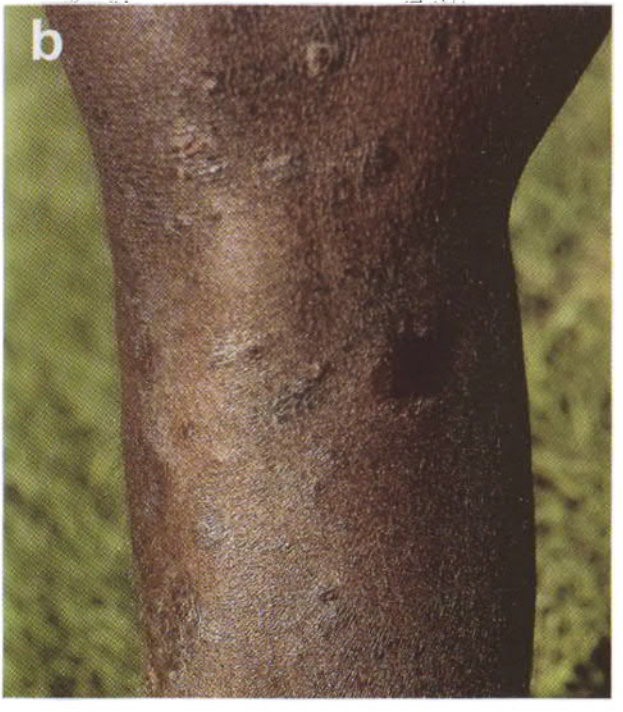
Making the diagnosis

- History is most important;
- May extract hairs or other material from a lesion for microscopic identification (e.g. using adhesive tape);
- Pricking the skin with the bindi-eye seed reproduces the clinical lesion in bindi-eye dermatitis;
- May need to distinguish from folliculitis. These lesions centre on a hair follicle so hairs may be seen emerging from some papules.

Significance

- Variable ranging from mild and self-limiting to persistent and requiring active treatment.





a *Bindi-eye sores of the elbows, which generally occur on the palms, soles, elbows and knees of children. Usually appearing in late spring and summer as grouped erythematous papules and papulopustules, a puncture site may be evident, may be smooth or scaly. May persist for months due to penetration of the skin by a hairy spine on each seed of* Soliva pterosperma *(bindi-eye weed).*

b *Biting midge (sandfly) bites on the leg.*

Infections: Viral

VIRAL WARTS

Summary: Benign papillomavirus infections most commonly affect children and disappear spontaneously, although can persist for years.

CLINICAL DESCRIPTION

- Solitary or multiple when often clustered or linear;
- Variety of clinical presentations
  - Verruca plantaris ('papilloma') on sole of foot; usually grow inward (endophytic) Also see differential diagnosis of callus of foot (page 126);
  - Verruca vulgaris (common wart) – cauliflower-like, raised (exophytic), keratotic, skin-coloured, may be solitary or multiple, especially on the hands;
  - Verruca plana (plane wart) – flat-topped, small, skin-coloured, usually multiple, clustered and often linear. Face and backs of hands most common sites, predominantly occurs in children;
  - Filiform wart – narrow base (pedicle), frond-like keratotic tips, usually solitary, usually seen around nose and eyes.

Epidemiology

- Most common in children but adults can be affected;
- Very common and numerous in immunocompromised patients.

Causes

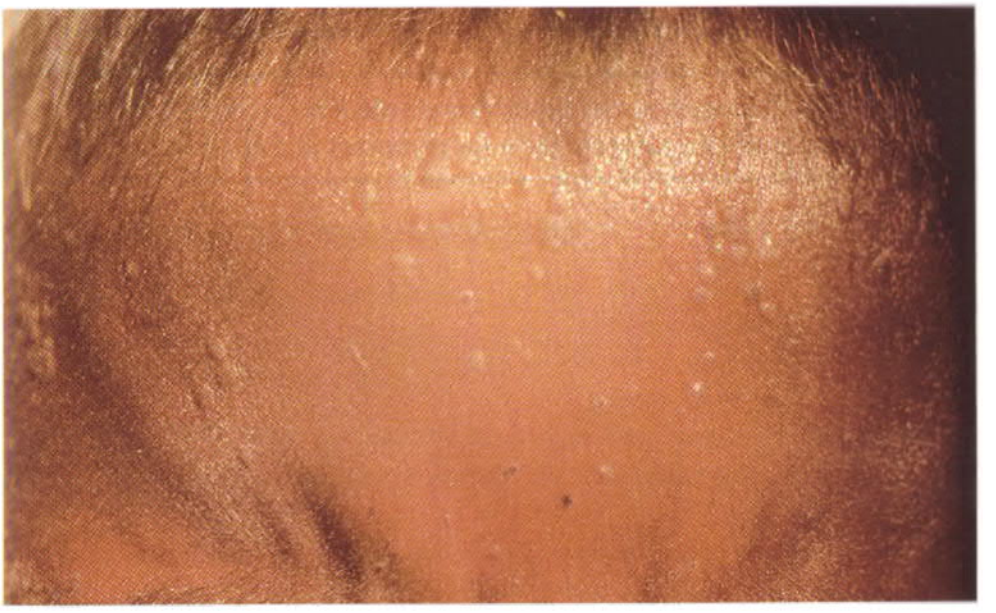
- Human papillomavirus (HPV) infections – many types;
- Of human origin, not caught from animals.

Making the diagnosis

- Usually clinical;
- Skin lines do not continue through a wart;
- On paring down, papillary capillaries become apparent as black or red pinpoint dots, compared to callosities (see page 126);
- Histology useful if solitary and need to distinguish from squamous cell carcinomas (which are rare in Aboriginal Australians) – hyperkeratosis, papillomatosis, parakeratosis, cytopathic effects in keratinocytes, acanthosis, prominent papillary capillaries.

Significance

- Benign and self-limiting but can cause social embarrassment;
- If particularly numerous and persistent may indicate individual is immunocompromised.



Clustered plane warts on the forehead.

MOLLUSCUM CONTAGIOSUM

Summary: Benign pox virus infection of the skin producing skin-coloured papules which resolve without treatment within months.

CLINICAL DESCRIPTION

- Solitary or more usually multiple, clustered, may be linear;
- Skin coloured papules;
- Variable size from 1 mm to 10 mm;
- Enlarge over 6–12 weeks;
- Central depression/umbilication/dell/dimple;
- May become red and inflamed shortly before disappearing spontaneously;
- Any body site – begins where the virus is first inoculated (e.g. face or arms in children or the lower abdomen in adults). Genital lesions in children may be due to auto-inoculation by scratching or sometimes sexual abuse;
- 'Kissing lesions' across a flexure due to auto-inoculation;
- Duration of individual lesions and infection varies – usually within months but can persist up to 5 years;
- May heal with scarring.

Epidemiology

- Most common in children, spread by touch;
- In adults can be sexually transmitted - lesions then usually on lower abdomen;
- Subsequent spread by auto-inoculation;
- Spread said to be more common in hot climates where dress is light;
- Sharing of the bath, towels, bath flannels (face washers) promotes spread.

Cause

- Molluscum contagiosum virus (molluscipox) is a member of the pox family of viruses (Poxviridae), and is the largest human virus. There are two types: MCV 1 and 2.

Making the diagnosis

- Usually clinical;
- Extrusion of material from central umbilication spread onto a glass microscope slide and examined microscopically unstained, will demonstrate the typical molluscum bodies;
- Punch biopsy/excision (usually of a solitary persistent or rapidly growing papulonodule) for histology will distinguish from other solitary tumours; crateriform tumour with cytopathic effect beginning in stratum malpighii, and from molluscum bodies in centre of lesion, central opening.

Significance

- Cosmetic – parents often concerned by spread and persistence.

Solitary molluscum contagiosum showing central umbilication.

Infections: Deep and Systemic Mycoses

Summary: A number of unusual yeast and fungal infections can be acquired from the environment resulting in persistent skin swellings, some of which are associated with systemic illness and, although uncommon, are important to consider.

CLINICAL DESCRIPTION

- Generally chronic and run a prolonged course over several years; diagnosis is often delayed; treatment frequently disappointing; may cause ill-health and disability or death.

1 Cryptococcosis

- Synonym: torulosis;
- Due to the environmental yeast *Cryptococcus neoformans* var. *gattii*, which is associated with river red gums;
- Not rare, most common of the primary deep mycoses reported;
- Primary cutaneous disease follows trauma and inoculation;
- Skin may be involved by dissemination from meningitis or pulmonary infection – skin lesion(s) may be the first clinical presentation;
- *Cryptococcus neoformans* var. *gattii* has also been reported to cause a primary cryptococcal cellulitis.

2 Mycetoma

- Synonyms: Madura foot, maduromycosis;
- Due to *Nocardia, Madurella, Aspergillus* and other environmental fungi;
- Numerous but localised skin nodules or tumour-like mass, swelling, sinuses discharging serosanguinous fluid, subcutaneous abscesses;
- May involve fascia and bone;
- Chronic;
- Failure to respond to numerous courses of antibiotics;
- Perhaps a history of penetrating skin injury – causative organisms are usually associated with soil or decaying vegetation;
- Most commonly reported from tropical and subtropical regions. Reported but rare in Aboriginal Australians.

3 Sporotrichosis

- Due to the dimorphic fungus *Sporothrix schenckii*;
- Nodular lesion(s), may suppurate and ulcerate, may remain fixed (single or multiple primary lesions), lymphocutaneous form – linear pattern along a lymphatic (sporotrichoid spread, see fig.b page 111), lymphangitis may occur, cutaneous dissemination rare, extracutaneous disease may involve bone, muscle, joints, lungs, central nervous system, genitourinary tract;
- Organism associated with soil, timber, bushes and decaying vegetation;
- Has been reported following ritual tattooing.

4 Chromoblastomycosis

- Synonym: chromomycosis;
- Various causative fungi including *Phialophora, Cladosporium*, which are isolated from timber and soil;
- Chronic, slowly progressive;
- Usually affects skin and subcutaneous tissues of the legs and feet;
- Localised;
- Lesions may be linear along lymphatics (sporotrichoid spread);
- Usually follows minor injuries such as puncture wounds and splinters from timber;
- Endemic in parts of north Queensland.

5 Actinomycosis

- Fungus *Actinomyces israelli*;
- Nodular skin lesions which breakdown and discharge a glairy type of pus, in which can be seen at times the so-called sulfur granules;
- Untreated these lesions form sinuses and persist indefinitely;
- Usually the lesions form slowly, are not very painful and at the beginning may appear like a subacute pustular infection of the deeper layer of the skin;
- Most common site is neck (oropharyngeal) but can occur anywhere.

Epidemiology

- Environmental infections, not spread person-to-person;
- More common in those who go bare-foot.

Causes

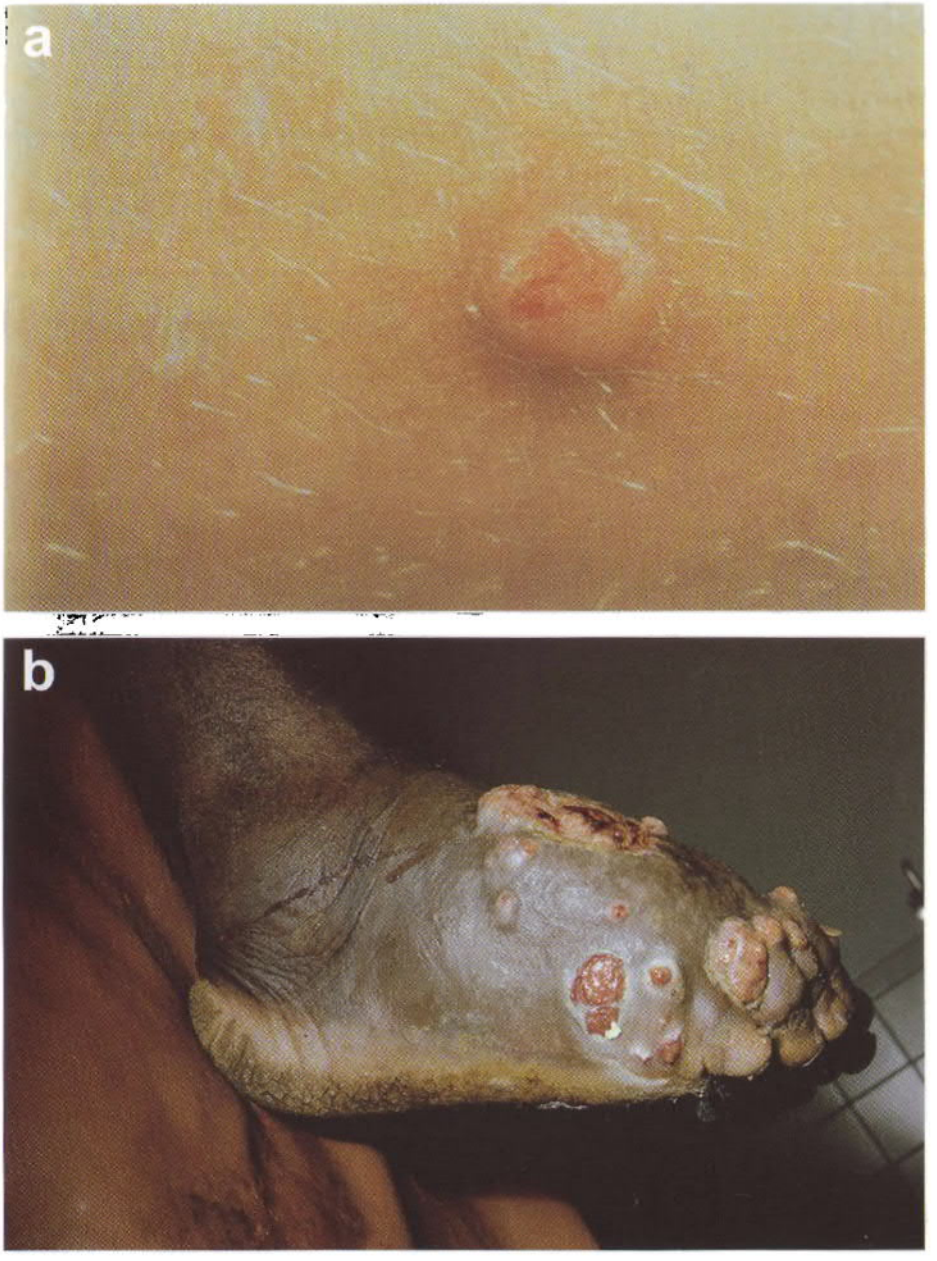
- Environmental organisms – soil, timber, vegetation;
- Usually follow penetrating trauma, so lesions begin on exposed site (e.g. foot)

Making the diagnosis

- Requires a high index of suspicion and repeated investigations (including skin biopsies for histology with special stains and culture for unusual organisms) to make the diagnosis in some cases.

Significance

- Some cause general ill health, disability and death, and so are important to recognise.



a *Cryptococcosis presenting as a skin nodule.*

b *Nocardiosis of the foot. (Reprinted with permission from the late Dr J Hawkins, Alice Springs Hospital, Alice Springs, Northern Territory, Australia).*

Bush Feet

Synonym: Desert feet.

Summary: Dried mud appearance over the tops of the feet in children going barefoot, particularly in desert regions. Is of no clinical significance.

CLINICAL DESCRIPTION

- Involves tops (dorsum) of toes and feet, front and sides of ankles;
- Bilateral, symmetrical, diffuse thickening;
- Colour is often that of the local sand, clay or mud;
- Progressive stages
  - Early – increased prominence of skin lines and slight roughness;
  - Progresses to resemble crazy paving;
  - Well-developed late stage – dried, fissured miniature mud-pan in appearance and texture.
- Onset soon after child begins to walk;
- Reversible – regresses with the wearing of protective footwear.

Epidemiology

- Common in those who go barefoot – almost universal up to puberty in central and northern Australia;
- Males and females;
- Childhood and adolescence 3–16 years.

Causes

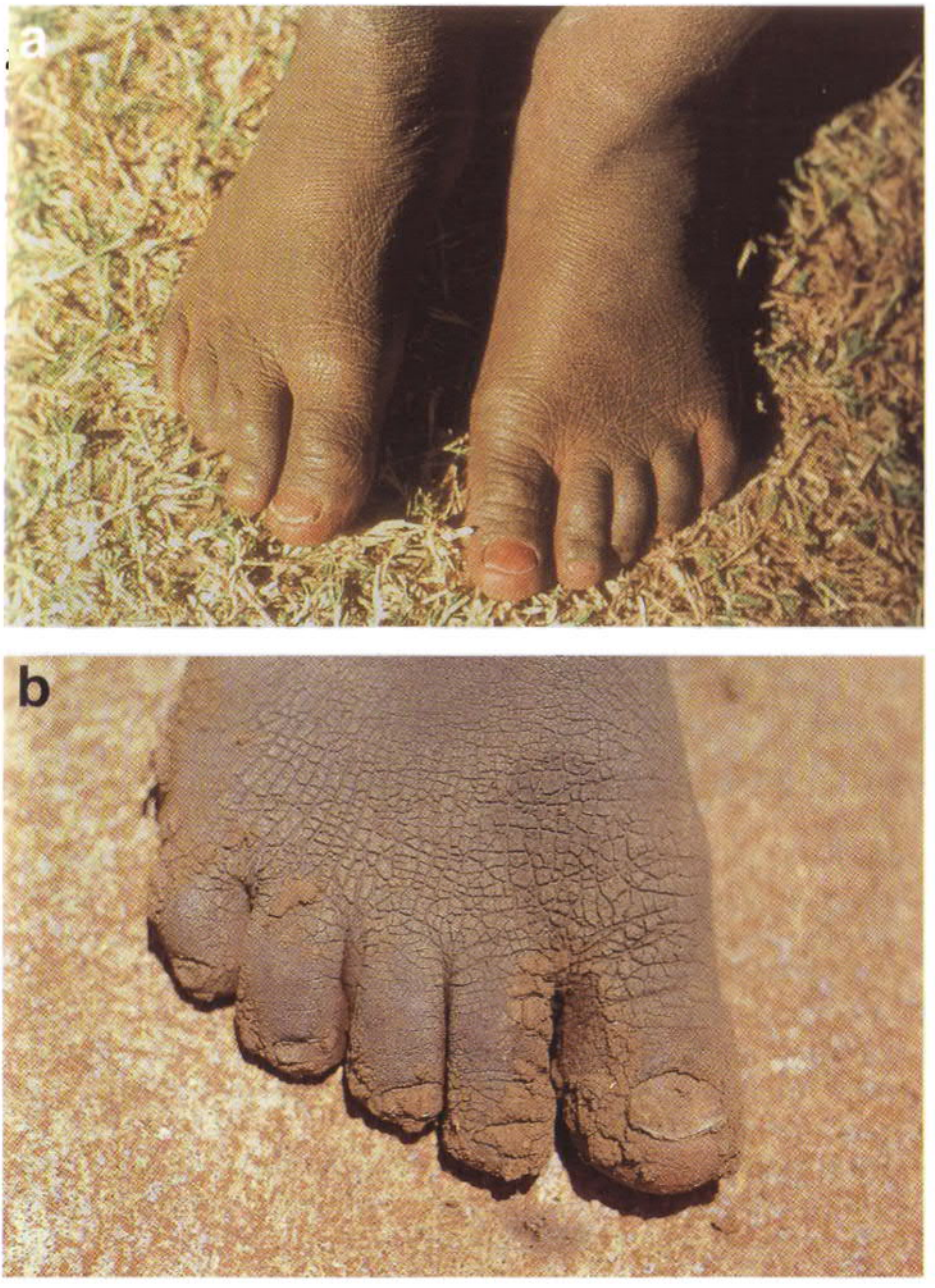
- Going barefoot in Australian bush;
- Reactive thickening of the outer horny layer of the skin (hyperkeratosis) from the repeated abrasive action of scrub, sand, clay and other soils.

Making the diagnosis

- Clinical;
- Distinguish from dried mud – will not wash off;
- Distinguish from ichthyosis – does not improve with moisturisers (emollients).

Significance

- None.



a *Early stage of bush feet showing accentuation of skin markings.*
b *Combination of desert feet and dried mud – late stage.*

Hyperkeratotic Soles and Callosities

Summary: Thickening of the sole of the foot, either diffuse or localised, usually as a reaction to pressure but may indicate peripheral neuropathy.

CLINICAL DESCRIPTION

- Diffusely thickened scaly soles of feet;
- May crack;
- Localised callus at pressure sites only;
- The hands and feet in leprosy become hard and callused, may crack and ulcerate.

Epidemiology

•. Common in those who go barefoot.

Causes

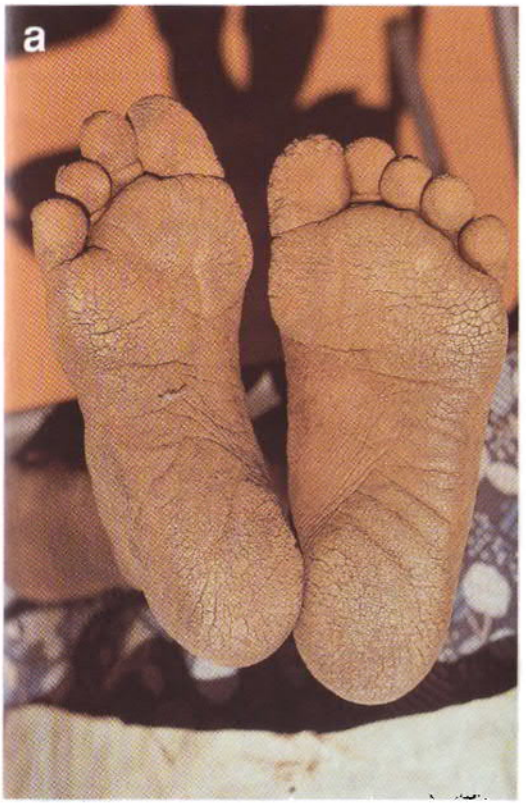
- Reaction pattern to pressure;
- In leprosy also due to failure to sweat.

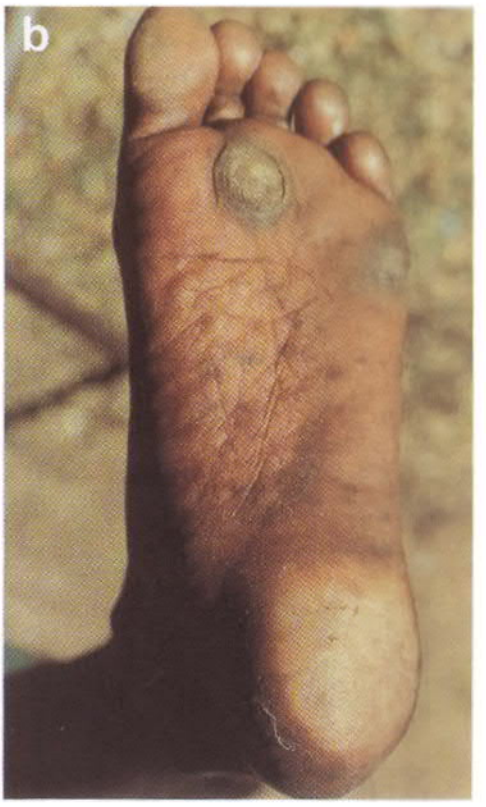
Making the diagnosis

- Clinical;
- May need to distinguish callosities from viral plantar warts – callosities occur only at pressure sites, show accentuation of skin markings and show featureless hyperkeratosis on paring without the black dots of papillary capillaries seen in warts (see page 116);
- Callosity may hide a neuropathic ulcer;
- Hyperkeratosis of the soles may need to distinguish from tinea pedis (ringworm of the foot, athlete's foot) by skin scrapings for fungal microscopy and culture;
- Crusted scabies can present with hyperkeratotic palms and soles – look for clinical features elsewhere and take scrapings for microscopy to demonstrate the mite, eggs and faecal pellets.

Significance

- None but associated disorders need to be excluded.





a *Hyperkeratosis of the soles.*
b *Callosities of the soles.*

Neuropathic Ulcers

Summary: Painless ulcers developing in the setting of a peripheral neuropathy and a cause of significant long-term disability.

CLINICAL DESCRIPTION

- Painless ulcers in numb hands and feet;
- Most commonly on the feet but also on the hands;
- May follow minor trauma of which the patient is unaware at the time (e.g. ill-fitting shoe, a stone in the shoe, barefoot, hot water, cigarette butt, campfire);
- Secondary infection common;
- Slow to heal;
- Heal with scarring/callosity;
- Re-ulceration of hard scar tissue;
- May be complicated by osteomyelitis;
- Commonest sites on the feet are under the heel, under the metatarsal heads, tips of toes and middle of the outer border of the sole.

Epidemiology

- Mainly in those areas where leprosy and its aftermath(s) remain.

Causes

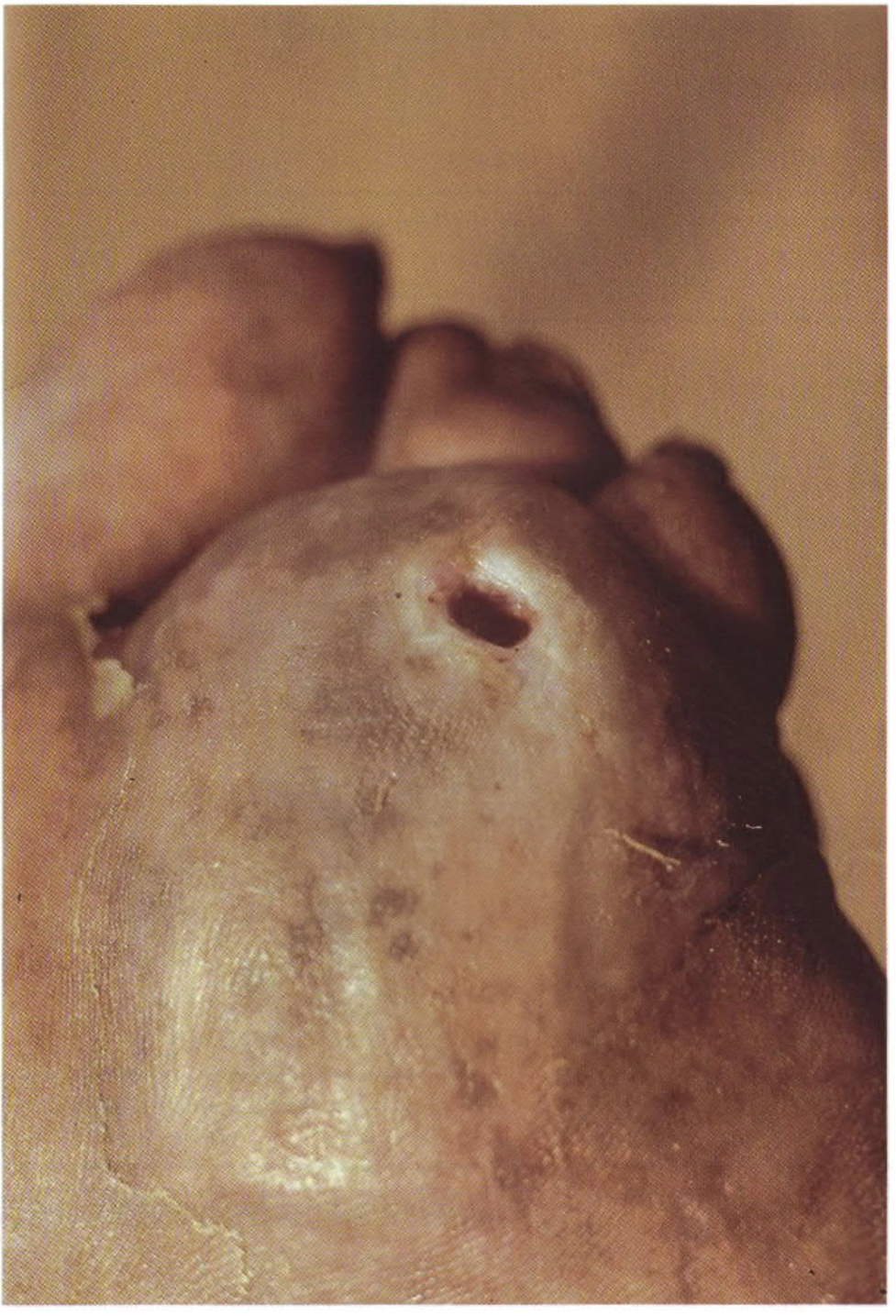
- Diabetes mellitus;
- Alcoholic polyneuritis;
- Leprosy;
- Other peripheral nerve lesions (e.g. post-traumatic).

Making the diagnosis

- History and general examination (e.g. for other features of diabetes or tuberculoid leprosy);
- Test for altered sensation and sweating;
- Blood glucose level;
- Nerve conduction studies;
- Swabs for bacteriology for secondary infection.

Significance

- Can result in significant long-term disability, particularly in remote areas where support services such as orthotists and rehabilitation programs are limited.



Consider a neuropathic ulcer.
See also Fig.d, page 105.

Fungal Infections of the Feet and Nails

TINEA PEDIS

Synonyms: Athlete's foot, ringworm of the foot.

Summary: Fungal infection of the feet usually presents as an asymmetrical, asymptomatic scaling but can spread to other body sites or be the portal of entry for bacteria resulting in cellulitis of the leg.

CLINICAL DESCRIPTION

- Asymmetrical, can be bilateral;
- Scaly accentuation of skin markings;
- Scaling or maceration (soggy white) between the toes;
- Can be blistering (vesiculobullous), inflammatory;
- Usually asymptomatic, may be itchy;
- May spread to other body sites;
- Less commonly, may see a similar presentation on the palms (tinea manuum).

Epidemiology

- Usually due to anthropophilic dermatophytes.

Cause

- *Epidermophyton floccosum* is commonest in Aboriginal populations.

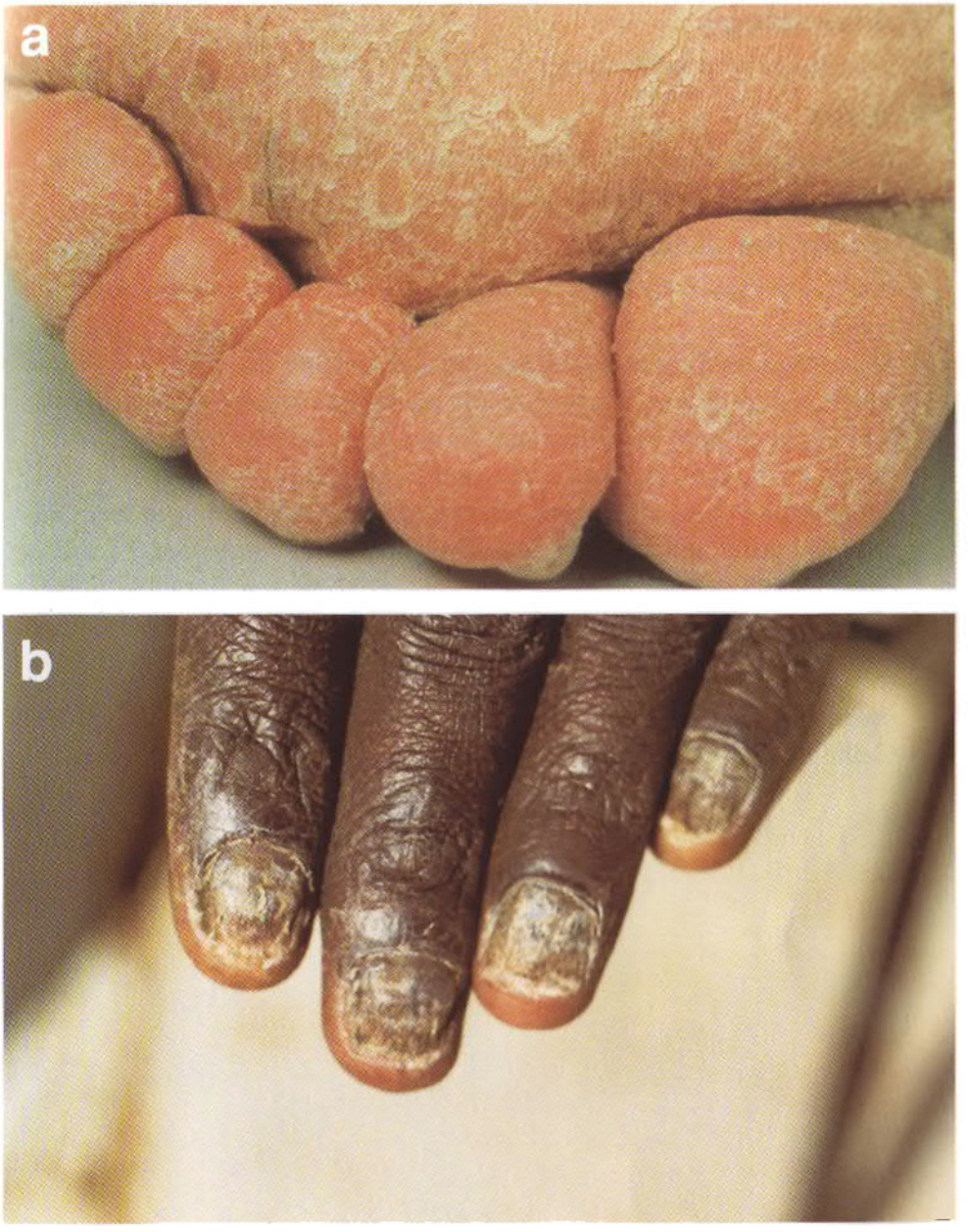
ONYCHOMYCOSIS

Synonyms: Tinea/ringworm of the nail, tinea unguium.

Summary: Asymmetrical thickening or other irregularity of nails due to a fungal infection.

CLINICAL DESCRIPTION

- Dystrophic, thickened, white, lifted up (onycholysis) nails;
- Chalky material beneath the nail (subungual);
- Asymmetrical – can be bilateral or unilateral;
- Finger and/or toe nails may be affected – toenails most common;
- May be associated with ringworm/tinea elsewhere on the skin.



a *Tinea pedis due to* Epidermophyton floccosum.
b *Onychomycosis due to* Trichophyton rubrum.

Epidemiology

- Usually anthropophilic fungi.

Causes

- In Aboriginal populations the most common causes are *Trichophyton rubrum* and *Trichophyton tonsurans*.

Making the diagnosis

- Nail clippings of affected parts of nails for fungal microscopy and culture;
- Skin scrapings for fungal microscopy and culture;
- May need to distinguish from traumatic nail dystrophy, particularly in the setting of peripheral vascular disease (e.g. diabetes mellitus), lichen planus of the nails and other rare nail dystrophies;
- Soggy white appearance between the toes can also be due to retained sweat or water and not infection – requires treatment to reduce risk of cellulitis.

Significance

- Infection can spread to other body sites (i.e. body, groin, face, etc.) usually via towels or clothing;
- Cellulitis may complicate tinea pedis especially if between the toes.

Section 3

Tables of Differential Diagnosis

TABLE 1 Classification of skin conditions by aetiology

A CHROMOSOMAL, DEVELOPMENTAL
Gyrate skin Folded skin of forehead Scalp
Hairy ears
Hirsutism
Alopecia and androgenetic (male pattern)
Madarosis, familial
Mongolian spot
Dermatosis papulosa nigra
Pseudo–acanthosis nigricans
Keloid
Pigmentation Gums Palms
Albinism
Neurofibromatosis
Naevus Blue Macular pigmented Hairy Melanocytic (moles) Macular hypopigmented Halo Infantile haemangioma (strawberry naevus) Epidermal (e.g. warty/linear)
Accessory nipple and areola

B CULTURALLY RELATED CONDITIONS

Tribal scars

Males	– Over chest and upper abdomen
	– Over deltoid
Females	– Upper back
	– Over deltoid

Avulsed upper incisor

'Nice marks'

Nose holes

'Sorry cuts'

Males	– Outer arm
	– Outer thigh
Females	– Scalp
	– Over hip joints
	– Calves

Circumcision

Subincision

Venesection

Burns (flaming ceremony)

Treatment scars

- forehead
- back
- lumbar region

Mucositis, chewing tobacco

Lateral malleolar bursitis

Tattoos

Children	– Various methods: plants, matches, razor blades
Prison type	

Ochre

Punishment 'payback'

C PHYSICAL CAUSES OF INJURY AND DISEASE

Hazards of the living environment
- Fire, broken glass, old iron, barbed wire, dogs, axes, spears and clubs
- No water, no food storage, no electricity and all that goes with it, hazardous sewage and garbage disposal (role of camp dogs)
- Alcohol, gambling, money, fights, old cars

Bush feet

Dry skin

Calluses

Hyperkeratotic soles

Sunburn

Acute – Infants and children
– Adults

Chronic

Photosensitivity

Miliaria

Trauma
- Various types of injuries such as abrasions, lacerations, penetrating wounds (e.g. knives, spears, gunshot); simple and compound fractures; burns; corns; calluses (see also culturally related conditions)
- Boomerang injuries (e.g. infected web injuries)
- 'Leopard skin'
- Burns – accidental, punishment, ceremonial

Bites and stings

Noxious and harmful plants

D CHEMICALS

Mottled teeth due to natural fluoride in water

Drug reactions

Kava dermopathy

E BIOLOGICAL AGENTS

Viral

- Warts – Plane, common, filiform, plantar
- Molluscum contagiosum
- Herpes simplex (cold sore, genital herpes)
- Herpes varicella zoster – chickenpox, shingles
- Measles
- Rubella

Bacterial

- Boils (furuncles), carbuncles
- Impetigo
- Infected injuries
- Ecthyma
- Streptococcal and staphylococcal ulcers
- Trichomycosis axillaris
- Leprosy
- *Mycobacterium ulcerans*
- Yaws – apparently eradicated
- Syphilis
- Donovanosis
- Gonorrhoea

Fungi and yeasts

- Tinea (ringworm)
  - *Trichophyton rubrum* (granular variant)
  - *Trichophyton tonsurans*
  - *Trichophyton violaceum*
  - *Microsporum canis*
  - *Epidermophyton*
  - *Floccosum*
- Candidosis – *Candida albicans*
- Pityriasis versicolor
- Nocardiosis
- Sporotrichosis
- Cryptococcosis
- Chromoblastomycosis

Insects

- Scabies
- Pediculosis
  - Head lice
  - Pubic lice
- Myiasis

Bites and stings

Noxious and harmful plants

F NEW GROWTHS AND CYSTS	
Basal cell carcinoma	– rare/apparently absent
Squamous cell carcinoma	– rare, of lip in discoid lupus erythematosus, external ear, anal
Melanoma	– rare, of sole of foot
Ameloblastoma	
Skin tag (achrocordon)	
Pyogenic granuloma	
Dermatosis papulosa nigra	
Seborrhoeic keratosis	
Epulis	
Milial cyst	
Epidermal cyst (sebaceous cyst)	
Xanthomatosis	

G INFLAMMATORY DERMATOSES
Discoid lupus erythematosus
Acne
Dermatitis/eczema Atopic – rare Cradle cap Seborrhoeic dermatitis Blistering beetle/acid beetle Stasis
Psoriasis – apparently absent
Vitiligo – rare
Bullous pemphigoid – rare

H MISCELLANEOUS

Alopecia areata – apparently absent

Ichthyosis
- Congenital
- Follicular
- Acquired

Varicose veins and their skin sequelae – uncommon

Ulceration of the foot
- Traumatic
- Leprosy

Peripheral neuropathy
- Diabetes
- Alcohol

TABLE 2 Skin signs and common causes

GUMS, BUCCAL MUCOSA AND TEETH
Variable patterns of pigmentation – none, small patches, larger areas
Avulsion of an upper incisor – males in central Australia
Mottled teeth from fluoride in some areas
Cheek biting, chewing
Chewing tobacco mucositis
Focal epithelial hyperplasia
Leprosy, lepromatous mucosal papules

INCREASED HAIR
Hairy ears
Hirsutism – chin and lips of elderly women

HAIR LOSS ON THE SCALP – WITHOUT SCARRING
Tinea capitis – children and young adults *Trichophyton tonsurans, T. violaceum, Microsporum canis, T. rubrum*
Hair pulling among children in fights or play
Trichotillomania
Alopecia and androgenetic (male pattern)
Syphilis (secondary)
Diffuse alopecia in elderly females
Drugs
Alopecia areata (part–Aborigines)

HAIR LOSS ON THE SCALP – WITH SCARRING
After injuries
Burns
Sorry cuts – women
Kerion (inflammatory tinea capitis)
Discoid lupus erythematosus (DLE)

ACQUIRED HYPERPIGMENTATION

Melanocytic naevi and (moles)

Areas exposed to the sun

Pregnancy – face (melasma/chloasma), areolae, nipples, linea nigra, genitalia

Dermatosis papulosa nigra

Pseudo–acanthosis nigricans

Keloids

Tattoos

Post-traumatic – burns, injuries

During and after inflammatory conditions – healed ecthymatous sores, tinea (especially *T. rubrum*), DLE

HYPOPIGMENTATION AND DEPIGMENTATION

Generalised

- Albinism

Localised

- Birthmarks
- Perinasal and malar in some children
- Halo naevus
- Pityriasis versicolor
- Leprosy
- During and after inflammatory conditions – tinea, occasionally DLE when healed, Herpes zoster (shingles)
- Vitiligo
- After injuries – abrasions, lacerations, scars, some tattoos, burns
- After use of cryotherapy, cauterisation, hyfrecation and prolonged potent topical corticosteroid application

KELOID

- Decorative and tribal scars, ceremonial rituals
- Burns
- Jellyfish stings
- Lacerations and other wounds
- Medical – surgical incisions, biopsy scars, injections
- Acne – presternal region and back
- Discoid lupus erythematosus

LIP LESIONS

- Lips may be red, scaly, ulcerated, scarred, indurated or hypopigmented
- Habits – licked, sucked, bitten, chewed
- Injuries – in fights, at play, in traffic accidents
- Burns
- Mucositis – from chewing tobacco
- Discoid lupus erythematosus (DLE)
- Squamous cell carcinoma (SCC) in DLE and albinism
- Syphilitic chancres
- Actinic cheilitis (sun damage)

MADAROSIS – PARTIAL OR TOTAL LOSS OF EYEBROWS

- Familial (genetic)
- Traumatic – injuries, plucking, rubbing
- Burns
- Leprosy
- Syphilis – secondary
- Thyroid disease

MUTILATIONS AND SCARS

Cultural and tribal practices

Environmental injuries

Burns

Tattoos

Infections – ecthyma, leprosy, yaws, donovanosis, DLE, syphilis, *Mycobacterium ulcerans*

SORES AND ULCERS

Secondary infection of established lesions is common and often so gross that the underlying condition is masked. Examples include:

Injuries of all kinds

Bites and stings

Burns

Scabies

Head lice (pediculosis)

Ecthyma

Impetigo

Staphylococcal ulcers

Desert sore (Barcoo rot)

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Dr M Cook, Adelaide, South Australia.

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Dr Green used a German-made Pentacon single lens reflex shutter camera. It was manually operated, had a focal plane shutter and a 2.8mm Biotar lens. The film of choice was 35mm daylight type Kodachrome ASA 25. Most of the photographs were taken in natural light. All care has been taken to acknowledge sources of clinical photographs not taken by Allen Green.

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